THE NERVOUS COMPLICATIONS OF MALARIA.

by

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M.D. 1920.

NOTE: With three exceptions, all the cases in this series were under the care of the writer, and the clinical histories compiled by him.

CONTENTS.

Introduction.

A general survey of the literature relating to malaria conveys the impression that nervous disorders occurring in or after this fever are relatively infrequent, and that apart from the nervous perturbations of pernicious cerebral types, affections of the cerebrospinal axis are rare. To a certain extent this remark is equally applicable to the ordinary specific fevers which are observed in this country. It is only in recent years that attention has been directed to the rich harvest of symptoms on the nervous side which are ready for investigation. Nor is it surprising that nervous complications and sequelae should often escape adequate recognition, since in civil practice it is usually the physician in charge of fever hospitals, or the general practitioner who undertakes their treatment, and the more obvious and intractable forms of nervous disorder are only seen by the neurologist when they have been established for some considerable period.

In military practice, however, where special departments exist, the medical officer is more favourably placed with regard to his facilities for studying disease in all its aspects; he can at any time obtain the expert help of specialists, or immediately transfer his patient to the care of those who have made a study of the particular complication which has arisen. In this way excellent opportunity is afforded for observing the rarer manifestations

of zymotic and other diseases.

In the Macedonian campaign of 1915 - 1919 malaria was the greatest of all causes of admission to hospital. It is probably no exaggeration to state that at least three fourths of the British Salonica Force were infected with this disease. During tne twelve months ending October 1918 there were no less than 1,970,600 total days sickness due to malaria, the minimum number of cases in hospital on any one day being 6,855. Even these figures, large as they are, fail to emphasise the huge incidence of malaria among the British troops, since they do not include cases under treatment in regimental aid-posts and Field ambulances.

In the autumn of 1917 a neurological department was opened in one of the Base hospitals to supply the urgent need for the special treatment of functional and organic nervous disease occurring among the troops. It was soon found that many of the symptoms presented on admission were causally related to, or aggrevated by a pre-existing or accompanying malarial infection. The majority of cases were examples of functional nervous disorder, but a certain percentage presented symptoms of organic nervous disease, and of these a small group appeared to be conditioned solely by the parasites of malaria. Although in comparison with the general incidence of malaria in Macedonia the number appeared to be extremely small, nervous complications were by no means rare. Many cases occurred before the neurological department came into being, and even after

its establishment, were evacuated directly to the United Kingdom form the various General hospitals. Moreover, the aural and ophthalmic specialists naturally saw the greater number of cases in which complications affected the sense organs.

The aetiology of nervous complications

In a general survey of the nervous complications and sequelae of malaria it would be out of place to devote much space to a description of the malaria parasites and their special mosquito-hosts.

I shall content myself by stating that the two commonest types were the simple benign tertian and the subtertian malarial fever. Mixed infections and benign quartan fever also occurred, but in a relatively small percentage of cases, and so far as I am aware nervous complications were never observed in quartan fever. During the early part of the malarial season the prevailing species of mosquito was Anopheles maculipennis. The appearance of malignant fever was associated with the same parasite and also Anopheles superpictus. Most of the admissions to hospital in the early summer (May to August) were cases of benign tertian fever, while in the autumn (Septembet to November) a very large number of cases of malignant subtertian fever were seen. Cases occurred, however, during every month of the year, and relapsing cases were quite numerous in the early months of the year. With regard to the cases presenting nervous complications, their incidence corr-

esponded to that of the uncomplicated fever, the majority of cases occurring during the malarial season.

The Parasite.

Owing to the routine practice of giving large doses of quinine to infected men before blood examination was performed, in quite a number of cases, no parasite could be demonstrated; in those cases in which they could be found the subtertian parasite (Laverania malariae) was the most frequent type. But occasionally the plasmodium vivax was reported to be present; it is of course possible that in such cases there was a mixed infection for it would be unlikely that an Army bacteriologist having found one type of parasite, would continue to search in the hope of finding others.

In 26 patients presenting nervous complications the benign tertian parasite was reported to be present in 7 cases, the malignant tertian parasite in 5 cases in 1 case there was a mixed infection with both B.T and M.T. parasites, and in the remaining 13 cases blood examination was negative.

Contributory factors.

Influence of Age. Age did not appear to be an important etiological factor, but no definite statement on this point is permissable, as all the observed cases of nervous disease were among soldiers of varying ages determined by military requirements, and up to the present no tables have been published showing the age incidence of uncomplicated malaria.

Fatigue.

In four patients fatigue appeared to be related to

the localisation of the nervous sequelae.

Thus, a soldier in the mechanical transport section of the R.A.S.C. had been employed in wielding a heavy hammer some days prior to the onset of his malarial attack. During convalescence he developed a musculo-spiral paralysis in the limb which had held the hammer. In another instance peripheral neuritis attacked the lower limbs of an officer who had spent three days in Athens, and had walked long distances along rough roads and tracks.

Previous disease.

A history of syphilis was given in two cases, and two patients had suffered in infancy or childhood from diphtheria.

Previous attacks of malaria.

Of the 26 cases in this series 15 gave a history of previous attacks; in 9 the nervous complications followed a primary attack of malaria. In 2 cases information as to previous attacks was not forthcoming.

The severity of the malarial attack, and the effects of treatment.

Although Macedonian malaria was undoubtedly of a severe type it must be admitted that it was also a malaria badly treated, and it is therefore of interest to inquire whether nervous complications were relatively more frequent in patients therapeutically neglected. It would be unwise to form any definite conclusion from the small number of cases in this series, but it may be noted that in only 5 cases had the treatment been haphazard and

inadequate.

With regard to the severity of the fever preceding the nervous complication (and here we exclude six cases of cerebral malaria) the attack could be characterised as mild in 13 cases, as of ordinary severity in 7, and as severe in 6 cases.

The nature of the nervous complications

Neurotropism of the plasmodium.

It has long been recognised that the malignant parasite is peculiar in having a special predilection for the nervous system, and is responsible for nearly all cases of cerebral malaria. Indeed, some writers go so far as to suggest that all cases with involvement of the nervous system are infected with the subtertian parasite. The evidence at my disposal, however, strongly favours the view that nervous sequelae may complicate cases of benign tertain fever; in seven cases the plasmodium vivax was the only parasite found, and in each instance the paroxysms of fever occurred every third day. Nervous complications related to benign tertian fever have also been reported by other observors. In the Daily Review of the Foreign Press (1) reference is made to two cases recorded by Hesse. In his two cases the complications were of the cerebral type, and in one free tertian schizonts were found in the brain capillaries, while in the other the intermittent character of the temperature indicated that it was a case of chronic relapsing malaria.

Another case,abstracted in the same journal,was observed by Pyskowski(2); the onset was like that of cerebrospinal fever with a polymorphonuclear leucocytosis in the cerebrospinal fluid. Fourteen days later an attack of fever occurred, and tertian parasites were found in the blood.

Phear(3) mentions a case on a hospital ship simulating cerebrospinal meningitis in which blood examination showed a heavy infection with benign tertian parasites.

It is also interesting to note that in the Macedonian series to be presently described a very considerable number of cases (50%) were related to mild attacks of fever. In those cases in which the fever was of a severe type the nervous complications usually appeared early or at the beginning of an attack. On the other hand, where the paroxysm was of a mild character they usually occurred in convalescence or in an apyrexial period.

Clinical forms.

The clinical forms are numerous and varied, and the cases personally observed may be classified as follows:-

3 organic lesions.....1. Cerebral
2. Pseudobulbar
3. Cerebellar
4. Cranial Nerves
5. Spinal Cord
6. Polyneuritis
7. Neuritis

8. Herpes Zoster.

Cerebral Types.

Foremost among the graver complications of malaria must be placed "cerebral malaria" which was responsible for rapid death in a very large number of cases. Its symptoms are familiar enough, and it may be questioned whether there is justification for classifying it as a complication of malaria, for it occurred so frequently in Macedonia that it might be regarded rather as a clinical type of the disease. The writer had the opportunity of seeing a very large number of cases especially among soldiers of the Royal Serbian Army, but unfortunately at a period (the summer of 1916) when systematic observations could not be made owing to stress of work.

The following types may be recognised:-

1. The ordinary Comatose or Delirious type.

The symptoms of this type bear a close resemblance to those of meningitis or cerebral irritation. They may occur at any stage of the malarial attack, and vary much in character and degree. The three cardinal symptoms - headache, vomiting and shivering - may usher in the attack, or they may be absent. The temperature is extremely variable; it may reach hyperpyrexial levels or be sub-normal. A history was frequently obtained that the patient

was found in a confused and feverish state, and rapidly developed either acute delirium or loss of consciousness, which persisted for several days or unti death occurred. In others examples cerebral symptoms made their appearance at a period when the patient seemed to be in the initial stages of convalescence.

The following case may be cited:-

Sgt. Y., 10/Devon Rgt., aged 26, was admitted to hospital on August 28,1918, complaining of weakness, and of pain in the left side. He had had several attacks of malaria, the last being on August 13. On admission he was anaemic, debilitated, and had a moderate degree of enlargement of his spleen. On the afternoon of August 28 his temperature rose suddenly to 105°F. He had intense headache, and was irritable and restless. An intramuscular injection of 25 grains of quinine hydrochloride was given. Next morning he was observed to be somewhat confused, although still able to converse in a rational manner. Later in the day he became drowsy, in spite of a second intramuscular injection, and quinine intravenously. On the morning of Ausust 30 he was comatose, his respirations laboured and irregular, his pulse small and feeble, and his skin pale and clammy. He lay with flaccid limbs, and conjugate deviation of the eyes to the right was present; his temperature was subnormal. A blood film showed a mixed infection with the parasites

of benign and subtertian fever. He died at 6.15 p.m.

22. The fulminating type

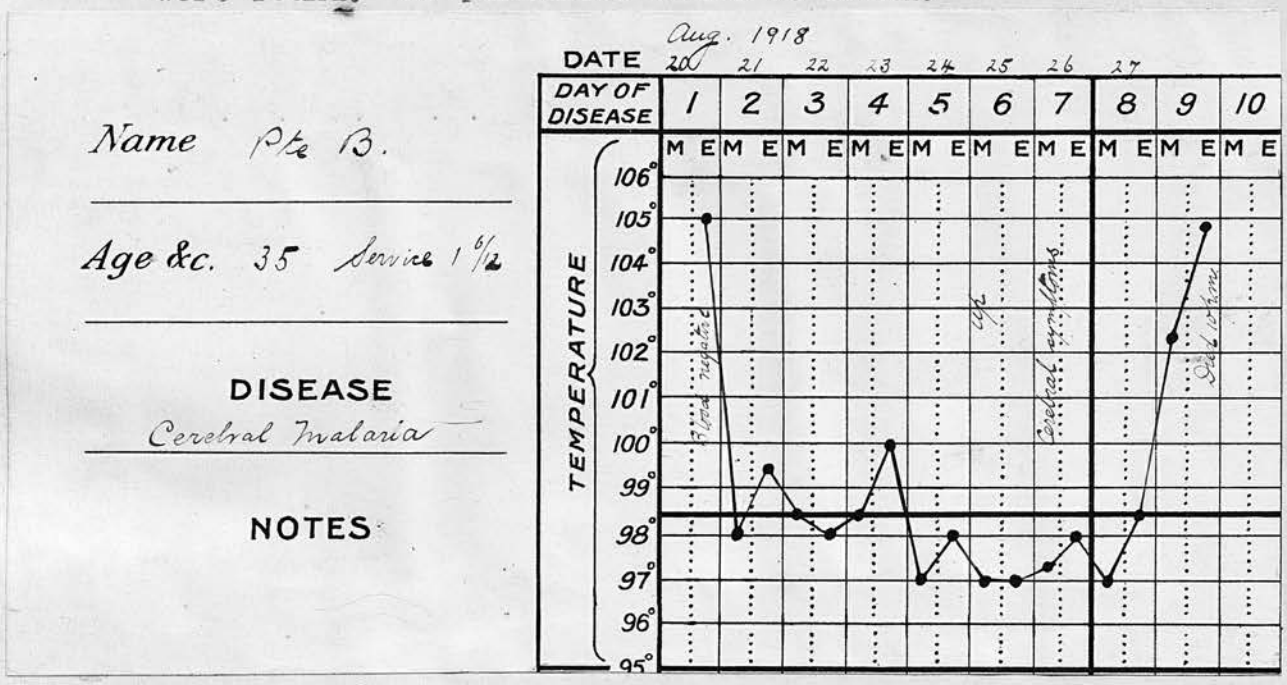
This type differs only from the foregoing in that the onset is extremely sudden, and the course one of great rapidity, a fatal termination usually occurring within 48 hours of the onset. Symptoms of intense poisoning or of collapse may be noted, and occasionally various forms of erythema or purpura may be noted.

The patient may appear to be quite well, and unexpectedly lapse into unconsciousness. The following case illustrates this type:-

Pte. B., R.A.M.C., aged 38, had a typical primary malarial attack on August 20, 1918. Blood examination was negative; his spleen was enlarged and palpable. He made satisfactory progress, and on the evening of August 25 went for a short walk, wrote several letters, and appeared to be in good spirits. Next morning at 5 a.m. when his temperature was taken he was rational and fully conscious. One hour later he was found in a semi-unconscious state, with dilated pupils, and irregular clonic movements of his limbs. At 9a.m. he was deeply comatose, his respirations being sterterous, his pulse rate 134, his temperature 96°F,

and his skin bathed in a cold sweat. He showed generalised muscular rigidity, and clonic movements, which were most marked on the right side. His tendon reflexes were increased, and his plantar reflexes of the extensor type; there was incontin-

ence of urine and faeces. Lumbar puncture yielded 20 c.c. of clear cerebrospinal fluid, which showed a moderate pleocytosis and a slight increase in globulin content. Blood examination was negative. Twenty four hours later (Aug. 28) the muscular rigidity gave place to flaccidity, and all reflexes were lost. In spite of energetic treatment the patient died at 10 p.m. on August 28. At the autopsy held 12 hours after death the macroscopic appearances characteristic of cerebral malaria were found. No parasites could be found.



Epileptiform Type.

This type is undoubtedly somewhat rare, and is said to be most frequently seen in children or young adults. The following case may be quoted as an interesting example, and is recorded in full as the neurological findings were of unusual interest.

Dvr. H. R.F.A., aged 20, had a primary attack of malaria in July 1917, and a number of relapses in

the autumn and winter, the last occurring on November 26. He was discharged to a convalescent camp on December 7, and readmitted to the Neurological Department on December 17 with the accompanying notes:-

"Was brought into hospital marquee at 6.30 p.m. by men in camp who state that patient was waiting along with others when he appeared to fall forward in a fit. When examined he appeared a little dazed, but answered each question. When put to bed he was almost immediately attacked by another seizure. Heart sounds good. Frothing at mouth; pupils large: reflexes normal: no incontinence. Present attack has now lasted 1 hour, 40 minutes".

State on admission.

General appearance.

Patient is flushed; lies with eyes closed, but not habitually. Respirations increased, varying in amplitude and rhythm: expiration noisy. Attitude: lying on back. Right upper limb abducted at shoulder flexed acutely at elbow, wrist pronated, fingers extended, thumb flexed. Left upper limb widely abducted at shoulder, flexed at right angles at elbow slight extension wrist, fingers flexed, thumb extended. Lower limbs flexed at both hip and knee joints, and adducted. Plantar flexion at ankle joints; limbs are tonically maintained in above attitude.

Neurological examination.

Makes no response to loud stimuli. Pupils slightly unequal; right larger than left.

Eyes subject to slow synergic movements from side to side. Pupils react normally to light. Corneal reflexes present. No asymmetry of face; mouth appears to be full of saliva. Kernig's sign positive: Brudinski's sign positive.

Reflexes: abdominals absent. Plantars, extension with withdrawal of feet. Supinator, knee, and ankle jerks brisk and equal; ankle and patellar clonus elicited; incontinence of urine. Lumbar puncture performed. Blood film shows M.T. crescents.

Progress.

11 a.m. Temp. 99 F., pulse 108, respirations 30 per min. Slight cyanosis. Heart and lungs normal; spleen enlarged.

He is much more restless; right upper limb flexed at all joints; left upper limb extended at all joints except fingers, which are flexed.

Rotation of head to left causes:-

Extension right upper limb, flexion left upper limb.

Flexion left lower limb, extension right lower limb.

Rotation of head to right causes:-

Flexion right upper limb, extension left upper limb.

Extension left lower limb, flexion right lower limb.

11.30 a.m. Patient is often in "C.6" attitude. The left upper limb is extended at all joints except fingers; right upper limb flexed strongly at all joints. Head slightly extended and rotated to right; lower limbs both extended. A few minutes later, right lower limb flexed at all joints, and left lower limb extended at all joints.

Left plantar reflex now normal. Om flexing head, both lower limbs are drawn up. There is marked adduction of the thighs, the left often being crossed on the right. The patient is exceedingly restless, continually altering his posture.

Treatment: Quinine hydrochloride gr.15, intravenously.

2 p.m. Sleeping quietly.

3 p.m. Restless and moaning; temp. 102 F., pulse 134. Generalised clonic spasm.

6 p.m. Still unconscious; cannot be roused to take nourishment: incontinence of urine

Treatment: rectal salines: calomel gr.4. Counter-irritation back of neck, and ice pack on head.

19/12/17. 4 a.m. Slight return of consciousness. Can be roused to take a little nourishment, but does not speak

8.45 a.m. Unconsciousness less deep; can be roused by auditory stimuli, but resists actively when moved. Limbs still flexed: extensor plantar reflexes Incontinence of faeces.

3.30 p.m. Improvement: no rigidity; opens eyes in response to loud stimuli. Pulse 80, large volume.

5.30 p.m. Vomited once.

7. p.m. Has spoken - said "Oh dear". Pulse 76, regular. Pupils contracted. Grimacing. Left knee jerk not obtained. Appears much better.

10.30 p.m. Pulse normal; sleeping quietly.

20/12/17 4 a.m. Showed symptoms of collapse.

9 a.m. Conscious Left knee jerk still depressed.

Cerebrospinal fluid (removed on 18/12/17) contained, erythrocytes 800 per c.m.m.,lymphocytes 7 per c.m.m., and slight globulin increase. No growth in culture.

21/12/17 Fully conscious this morning; remembers all that happened just before he fell. States that he had intense headache, shivering and vomiting. Knee jerks both present; plantars- extension. Diffuse motor weakness; no individual movement impossible. Incontinence has ceased. Temperature normal Pulse 72 per minute.

26/12/17 Slight epileptiform seizure. Quite normal an hour later.

31/12/17 Progressing very satisfactorily; allowed up.

5/1/18 Except for generalised headache, weakness, and anaemia patient is normal.

The patient had two subsequent attacks of fever in January, unattended by nervous symptoms, and was ultimately discharged on March 11, 1918.

Comment.

This case not only affords a typical example of the epileptiform type of cerebral malaria, but also illustrates certain mechanisms of the central nervous system. First, the peculiar postures of the limbs during the period in which the patient was deeply unconscious resembled in a marked degree the reactions which have been observed in monkeys. Graham Brown (4) has shown that the main effect of stimulation of one red nucleus is a movement of

flexion in the arm of the same side, and a movement of extension in the opposite arm. One of the most constant attitudes assumed by our patient was one in which the right upper limb was in a state of maintained flexor contraction, while the left upper limb was extended at all joints except those of the fingers.

Secondly, conjugate automatic movements were observed, analagous in all respects to those induced experimentally in decerebrate animals. Thus, on rotating the patient's head slowly to the right, the right upper limb became tonically flexed, while the left became extended. On turning the head to the opposite side the attitude of the upper limbs became reversed.

Similar phenomena have been observed by Magnus and Klein, Marie and Foix,(5) in cerebral hemiplegia and diplegia, and by Rothfeld in a narcotised animal with intact nervous system. These facts demonstrate how exceedingly complex is the automatic functioning of the nervous system, and their interpretation is a matter of some difficulty. Since they are most typically seen in conditions reducing to a maximum the cortical control, one may venture to suggest that in our patient the malaria toxin, by poisoning the higher cerebral centres, allowed these phenomena, latent in the normal state, to become manifest owing to liberation of the lower midbrain centres.

Cerebral Malaria in Benign Tertian Fever.

The occurrence of cerebral symptoms in simple tertian fever is distinctly unusual, and for this reason the following brief record is made.

Dvr. B., A.S.C., aged 42, was admitted to hospital on August 16, 1918. There was a history of repeated attacks of fever.

On admission he was comatose and had incontinence of urine. Blood examination showed a heavy infection with the small rings of Benign tertian fever.

His state on 18th. August was as follows:-

Patient's posture and behaviour varies; sometimes he lies passive with eyes closed, and head turned to one side, usually the left. At other times he is most resistive, struggling on the bed, and making inarticulate sounds.

Pupils dilated: right larger than the left; reactions normal. Corneal reflexes normal. He opens his eyes in response to loud auditory stimuli, but does not reply to questions. A pain stimulus causes immediate withdrawal of lower limbs. There is no paralysis, but marked choreiform movements of face.

Kernig's sign positive on both sides. Tendancy to head retraction.

Reflexes: Abdominal reflexes absent. Plantars: flexion response. Knee and ankle jerks brisk. There is incontinence of urine.

On the morning of 18/8/18 consciousness was fully restored, and patient made an uneventful recovery.

Cerebral Malaria in Benign Tertian Fever.

4 HOUR CHART.

DISEASE.

Benign Tertian Malaria.

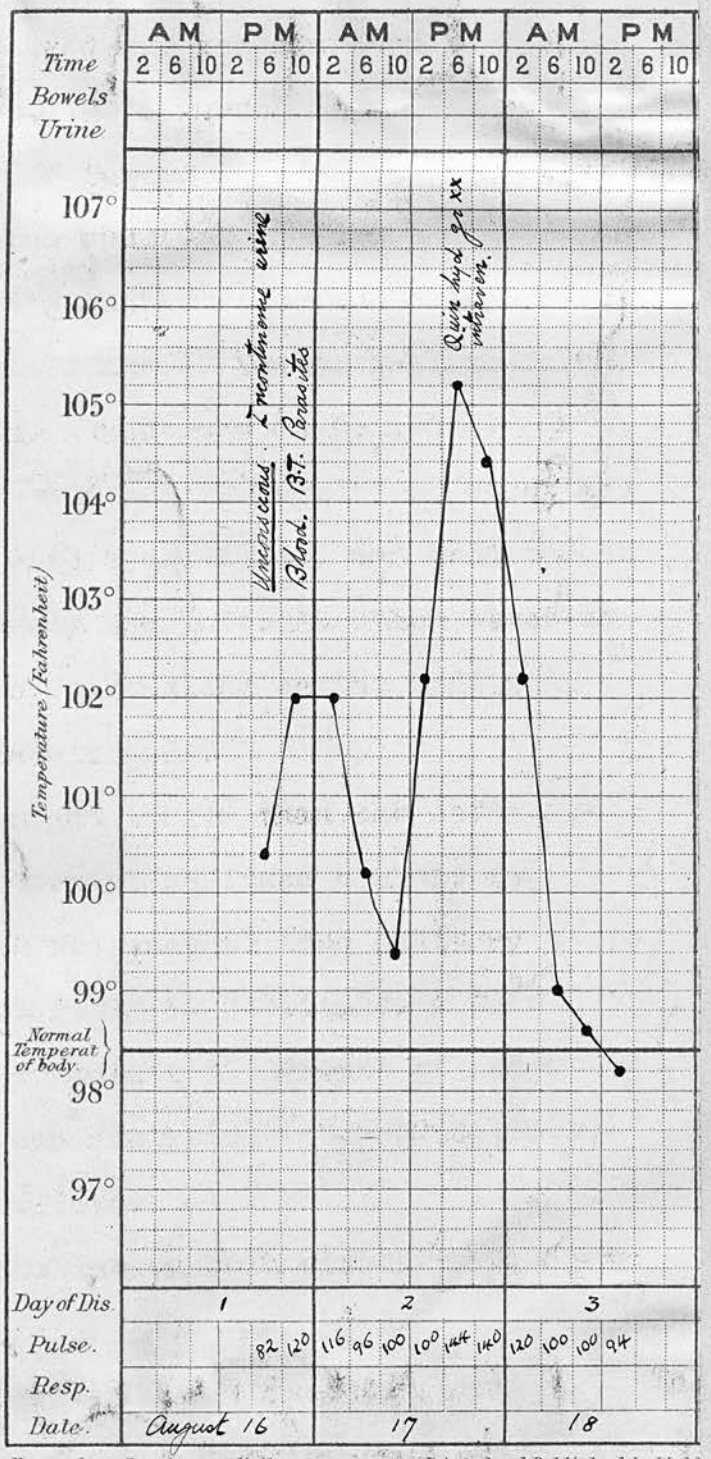
Name Pvt. B

Age 42

Diet

Case Book No.

Notes of Case



Date of admission 16. 8. 18.

Result

Entered at Stationers Hall. Printed and Published by Wood.

Pseudobulbar type.

Following the anatomical classification adopted above there has now to be considered a case, which as regards its etiology is probably unique.

Pte. G., R.A.S.C., aged 38, in civil life a farm labourer, was admitted to the neurological department on August 30, 1917. His family and previous history were negative.

His illness commenced on August 9 with headache, shivering and pains in the limbs. On the evening of the same day motor power in his lower limbs, and later his articulation, became impaired. He described his state as being like that of a drunken man - he could not keep on his feet, and had difficulty in comprehending spoken speech. There was also at first some difficulty in swallowing, and saliva dribbled from his lips. For several days he was very ill with high fever, and was admitted to a general hospital, where the following notes were made by Col. Purves Stewart on August 27. "Speech is normal; articulation very indistinct. Pupils and cranial nerves normal: no nystagmus. No cutaneous anaesthesia or analgesia. Gait: He takes feeble short steps and has difficulty in turning round. No weakness, tremor or ataxia of upper limbs. Slight weakness of lower limbs against resistance; no ataxia. Knee and ankle jerks brisk and equal: no ankle clonus. Plantar reflexes: both extensor responses. Abdominal reflexes absent.

State on admission 30/8/17.

Mental functions: memory impaired for recent events. Otherwise no abnormality; no emotional instability.

Articulation very much impaired.

Cranial nerves. Special senses normal. Face is somewhat expressionless. No difficulty in swallowing. Patient complains that saliva collects in his mouth. Tongue deviates very slightly to the right.

Sensory Functions. Everywhere normal; patient has no subjective sensations except "general weakness".

Motor functions: Upper limbs normal. Diffuse weakness in both lower extremities; no wasting.

Gait defective: he takes short steps and has difficulty in turning.

Reflexes: Abdominal reflexes absent, except right epigastric. Plantar reflexes: both extension. Tendon reflexes brisk and equal.

No parasites found in blood.

Progress.

1/9/17 Rigor and typical malarial attack last night. He seemed collapsed during the night. Both abdominal reflexes now present on the right side. Upper facial movements weak.

5/9/17 Temperature normal; feels much better.

21/9/17 Patient is now up. Articulation still defective.

27/9/17 All words appear equally difficult to pronounce. He is unable to close right eye by itself. When he closes left eye the levator anguli oris on the same side contracts. Tongue protrudes

evenly, and palate moves normally on phonation. Gait normal: no incoordination upper or lower limbs.

He was discharged on the 16th of October in good health. Two months later I had another opportunity of examining him. Apart from his articulatory defect which was still as bad, and his abnormal plantar reflexes, there were no neurological signs of importance.

Comment.

For the production of a pseudobulbar paralysis the centres or conducting tracts subserving the motor acts of articulation, phonation, mastication and deglutition must be affected on both sides, since the corresponding muscles must have a bilateral cortical innervation. The disease is usually of vascular origin, and a history is frequently obtained of successive apoplectic attacks. It is exceptional, as in this case, for a single and sudden attack to usher in the symptoms. An important diagnostic feature is the non-atrophic character of the paralysis, and another peculiarity is that the muscles which are deprived of voluntary control may be put into action by emotional, automatic or reflex means.

It must be noted in the above case that the symptoms were for the most part transient. Examination after the lapse of nine weeks showed that apart from dysarthria and an extensor plantar reflex on both sides little abnormality in the

nervous system could be detected. With regard to the site and character of the lesion but little can be said. The possibility that the pathological process involved the brain stem must be entertained, although the history of transient word deafness is rather in favour of a cortical origin; The suddenness of onset and the absence of clouding of consciousness are points worthy of note.

Cerebellar Types

The following four cases have many points in common and indicate clearly that the malaria parasite or its toxin is capable of causing disturbances of cerebellar function as grave as those exercised on the cerebral hemispheres.

Case1

Gnr. D., R.F.A., aged 29, was admitted to the Neurological department on August 12, 1917.

Family and previous history negative.

Present illness.

His illness commenced on July 11, 1917, with headache, vomiting and fever. At the onset he was working in the gun pit. He was admitted to a C.C.S. on the same day, where he developed diarrhoea and a temperature of 104 F. On the night of the 12th. he was delirious and fell out of bed. It was noticed that his articulation was indistinct, and that the finer movements of his limbs were lost. He was treated for hyperpyrexia and "cerebral Congestion" He improved, but two days later delirium again occurred, and on August 13 physical signs

of broncho-pneumonia made their appearance.
Three days later his temperature fell, and he became more rational, although his speech was still unintelligible, and the movements of his limbs incoordinate.

On August 3 he was seen by Col. Purves Stewart who made the following notes:-

"Understands and executes all verbal commands (i.e. no word deafness); promptly executes written requests (i.e. no word blindness); names promptly all objects shown to him, but articulation is jerky and indistinct. Writes accurately, but the script is slow, tremulous and small. Slight rotatory tremor of head at rest: also of angles of mouth. Irregular movements of protruded tongue. Coarse incoordination of upper limbs in finger-nose test; similar incoordination of lower limbs in heel-knee test. Pupils and cranial nerves normal. No nystagmus. No paralysis of trunk or limbs as he lies in bed. Well marked dysdiadochokinesis of both upper limbs. Also hypermetria with heel-knee test.
Spinal and abdominal muscles powerful: can sit up in bed without use of arms.
Reflexes: S.J., K.J.,and A.J. brisk and equal: pseudoankle clonus. Plantars: left flexor, right extensor. Abdominal reflexes brisk and equal.
Heart sounds clear and rapid. Patient is very emotional. Gait: when supported on both sides, walks unsteadily, trunk lagging behind lower limbs.
State on admission to Neur. Dept.

State on admission to Neur. Dept.

Patient lies quietly in bed; looks intelligent, and is well orientated in time and space; his memory and other higher mental functions are good. He is very emotional. He states that when his temperature fell to normal he noticed that he could neither speak nor move his hands properly, and had to be fed. His speech gradually came back, although when he wanted to say things he could not get the words out. Both upper extremities which were at first weak, gradually improved, the left more rapidly than the right. He never experienced difficulty in eating or drinking, provided that the food or cup was placed between his lips. He complained that his eyes "wandered a lot" - they seemed to leave the line of print before he had read it. He was unable to write.

Cranial nerves. Olfactory normal. Visual acuity normal; visual fields normal. Slight lateral nystagmus in extreme lateral positions, but sometimes appears to be present with eyes in any position. Pupils react normally to light and on accomadation. 5th., 8th., and 9th., cranial nerves normal. There is no difficulty in screwing up eyes or in showing teeth, but he cannot whistle, and his face looks featureless and bloated. There is coarse tremor of protruded tongue, the tip of which deviates slightly to the right. During examination slight rhythmic twitching of the angles of the mouth can be observed. Saliva constantly dribbles from his mouth.

Sensory functions. Normal

Motor functions. Motor power in the upper and lower limbs is good, and is equal on the two sides. There is marked hypotonia in the extensors of all four limbs.

Reflexes. Abdominal reflexes diminished on right side: normal on the left. Plantars: right, extensor: left, flexor. Tendon reflexes: upper limbs normal Knee jerks brisk and equal: ankle jerks normal: no clonus. Organic reflexes normal.

Coordination. In performing heel-knee test he shows marked dysmetria; this apparently is slightly greater if eyes are closed. Finger-nose test: if this is performed quickly, there is on the first attempt wild incoordination, the finger stabbing him all over the face until brought to rest. With repetition the movement becomes almost perfect; while present on both sides, this incoordination is most marked on the right. The test is less inaccurately done if performed slowly, and it is better in the vertical than in the horizontal plane.

There is an absence of the normal "rebound" when patient tries to flex elbow against observor, and latter lets go suddenly. Dysdiadochokinesis, dysmetria, and asynergia are marked. Thus, he is unable to make rapid alternate movements of pronation and supination, and in taking up a glass of water, or in dealing cards, he grossly overacts with his fingers. In attempting to throw

things the object is released from the hand too soon. Rombergism is present, and is uninfluenced by closure of the eyes. In walking his trunk remains passively behind, so that his legs appear to be running away from him.

The patient has difficulty in speaking, articulation being indistinct, and very explosive.

Progress.

A week later the incoordination was less marked, and he was just able to walk without assistance, his gait consisting of slow, short steps, with much swaying of the trunk.

22/8/17. The abnormal plantar reflex on the right side has disappeared.

25/8/17. There is present for the first time slight rhythmic involuntary movements of adduction and abduction in the right index finger. They occur only at the metacarpo-phalangeal joint. He can make rapid alternate movements of pronation and supination 120 times per minute on the left side, but only 66 times per minute on the right side.

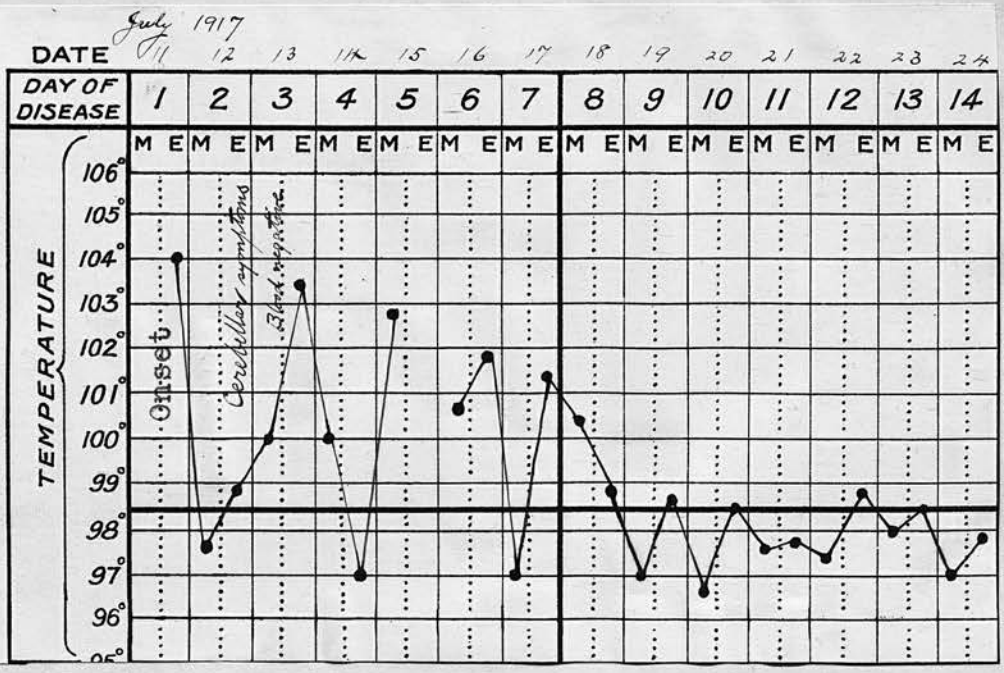
2/9/17 The rhythmic movements are now also seen in the right minimus.

15/9/17 The rhythmic movements are increasing gradually; today for the first time they appeared in the second and third metacarpals. They consist of flexion and extension on carpus, and are accompanied by the opposite movements (extension and flexion) at the metacarpo-phalangeal and inter-

phalangeal joints. The movement of minimus at metacarpo-phalangeal joint persists, but is still adduction-abduction. The movements are slighter at the proximal than at the metacarpo-phalangeal joint, and the movement at the distal interphalangeal joint is extremely small. Movements now also occur at the wrist joint, and there is some evidence of pronation and supination at the radio-ulnar joint. The rate of movement in minimus is 160 double phases per minute

16/9/17 Similar movements have now appeared in the left index finger.

During the month of October the involuntary movements persisted; the patient's gait markedly improved, and his articulation to a less extent. Pointing tests were almost accurate, if slowly performed. The patient was evacuated on Dec. 18 when it was noted that further improvement had occurred, and the involuntary movements had almost ceased.



ame
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DISEASE
Cerebellar Syndrome.

NOTES

Case 2.

Pte. E., 2/E. Kent Rgt., aged 21, had a primary a attack of malaria in the Spring of 1916; he had twelve subsequent attacks. The last occurred on November 14, and was of ordinary severity.

He made a good recovery and remained well until the afternoon of December 1, when he suddenly became shaky in the legs while marching to work.

Next morning he felt much worse, and noticed that his speech had become altered - he had to "force the words out". He felt giddy and walked like a drunken man, tending to fall forwards.

Soon after admission to a general hospital his articulation became more defective, and he experienced stabbing pain in the head and lower limbs.

During his evacuation to the Base he vomited, and noticed that he "could see more to the left than to the right". On December 6 he commenced to improve His articulation became less indistinct, and he essayed to walk, but without success. He was admitted to the Neurological department on Dec. 8.

State on admission.

Complaint: difficulty in walking and in speaking.

Higher mental functions. His general intelligence is fair. He understands written and spoken speech. His memory is fair.

Articulation: markedly affected; speech is explosive, dysmetric, and difficult to understand.

CranialnNerves. The special senses are normal, except that there is slight constriction of the

visual fields to the right. The left pupil is slightly larger than the right; both react normally to light and on accomadation. There is rotary nystagmus when the eyes are directed to the right. The sensory and motor functions of the fifth cranial nerve are normal

Facial: In smiling there seems to be more movement on the left than on the right side of the face. The tongue protrudes slightly to the right, but without tremor. The other cranial nerves are normal.

Sensory functions. Apart from hyperaesthesia of the calves on pressure, no abnormality can be detected.

Motor functions. The left upper limb is weaker than the right,and in it movements of extension are weaker than those of flexion. In the lower limbs movements of flexion are weak; there is distinct atonia of the left thigh and calf muscles. He lies with legs crossed, and if disturbed at once reassumes this attitude.

Coordination. In performing finger-nose test he shows a certain amount of incoordination in the left upper limb, and in the same limb there is dysmetria when he is asked to pick up small objects. There is no deviation in the pointing tests. He shows dysmetria in the left lower limb when performing the heel-knee test. There is no asynergia. Rombergism is present: he tends to fall backwards and to the right. Gait: markedly cerebellar in type: he walks with a wide base, keeping lower

limbs rigid, and reeling from side to side.
Dysdiadochokinesis is present in the left hand (opening and closing hand test).
Reflexes: Abdominal reflexes present and equal: p plantars, flexor response.
Tendon reflexes: Supinator reflexes equal; left B.J. and right T.J. exaggerated. Ankle jerks normal.
Organic reflexes normal
Progress.
12/12/17 Nystagmus less : Rombergism less; gait improving.
23/12/17 Rapid improvement, but no change in articulatory defect.
26/12/17 Slight malarial attack. Blood negative.
There is now no dysmetria, asynergia, dysdiadochokinesis, Rombergism or nystagmus. Tendon reflexes normal.
This patient was ultimately able to return to duty.

Case 3.
Pte. H., 1/Suffolk rgt., aged 23, had no illnesses until June 1916, when he had a primary attack of malaria, being one month in hospital. During 1917 he had relapses every few months, and on his ninth attack on 10/12/17 was again admitted to hospital. Diziness, sickness, and vomiting were complained of, and the patient stated that he had no control of his legs. He was slightly jaundiced and his spleen was palpable. Vomiting continued to be a troublesome feature until January 1, 1918.

The following notes were made by myself on Dec. 28
Complains of vomiting, diziness -"things go backwards and forwards"-, and of difficulty in walking. Mental functions normal; speech normal. Articulation slow and indistinct. Pupils dilated, equal, and react normally to light and on accomadation. No nystagmus. Stellwag's and Von Graefe's signs positive. Palate moves normally: tongue protruded straight.
Sensory functions normal
Motor functions: slight weakness hand grasps, and in lower limbs. There is incoordination in performing finger-nose test: patient stabs his cheek with his finger. Incoordination on performing heel-knee test, or on groping with either limb. Marked dysmetria of lower limbs: patient overshoots the patella by some inches on performing heel-knee test.
Gait: Patient cannot walk without support, and sways from side to side; he tends to fall forwards, and keeps his feet widely separated.
Muscle tonus of lower limbs diminished.
Reflexes: Abdominals normal; plantars: flexor response both sides. S.J.,K.J., and A.J. brisk and equal. Organic reflexes normal.
When the patient was transferred to the Neurological department on 21/2/18 he had greatly improved. Neurological examination was negative,save for the following: dysmetria right lower limb when performing heel-knee test. Gait: patient walks with feet separated, staggers to one or other side, and

has difficulty in turning.

Case 4.

The following case came under my care during the winter of 1919.

Sapper S. R.F.A., aged 19, was admitted to the R.V. Hospital, Netley on October 5, 1919.

Family history negative.

Previous history. No illnesses of importance in civil life, except convulsions in childhood.

He went to South Russia in 1919, and had a primary attack of malaria in July. His illness commenced with headache, shivering, pains in the limbs, and fever (temperature 104 F.). The parasites of malignant tertian fever were found in his blood on July 28. His spleen was easily palpable.

Two days later he became delirious, and on August 1 his temperature fell to normal. It was then found that he was in a condition of "aphasia", and passed urine and faeces involuntarily. On August 8 he developed a left sided parotitis, and was again unconscious for 13 days. The rings of plasmodium falciparum were present in his blood on August 26. He made a slow recovery, and was eventually evacuated to the United Kingdom.

State on Admission.

Complaint: difficulty in walking and speaking.

General Condition: pale and debilitated, with marked enlargement of spleen.

Neurological examination.

Higher mental functions.normal; there is marked

emotional overaction. No tremor or abnormal movements.

Speech normal. Articulation is slow, slightly slurring, explosive, and somewhat syllabic. He has especial difficulty with such words as "necessary" or "British".

Handwriting: he writes slowly and laboriously, individual letters being clumsily formed.

Pupils equal and regular in their outline: reactions to light and on accomadation normal. No nystagmus.

Cranial Nerves. Normal, except for paresis of muscles innervated by upper branch of left facial nerve (due to implication in operation scar).

Sensory functions normal

Motor functions. Motor power and tonus normal.

Gait: patient walks with trunk bent somewhat forwards, and head also bent; upper limbs adducted and held stiffly; he takes short steps, but does not deviate to either side, except in turning. As his face is rather expressionless and his brow wrinkled, his appearance when walking suggests to some extent paralysis agitans.

Coordination: No Rombergism.

Rebound phenomenon: absent right upper limb, present left.

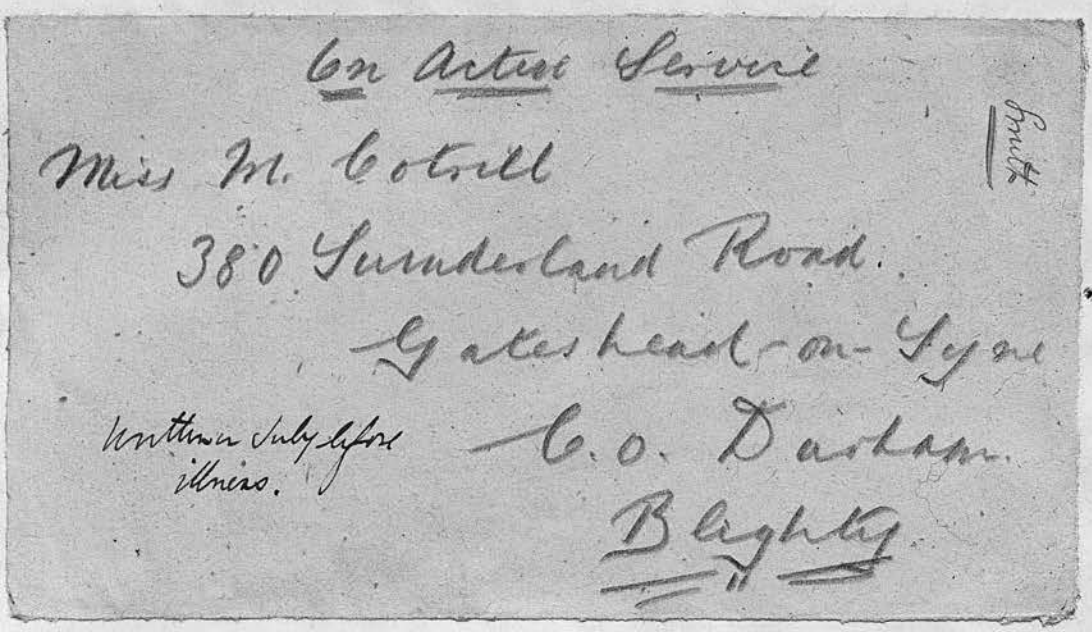
Cerebellar tests

1. Dysmetria Finger-nose test: slight dysmetria left upper limb. Picks up pin or glass of water with accuracy.

Heel-knee test: marked dysmetria with either limb;

Case 4. Sapper S. Cerebellar syndrome.

1. Specimen of handwriting before attack


On Active Service

Miss M. Cotsill

380 Sunderland Road.

Gateshead-on-Tyne

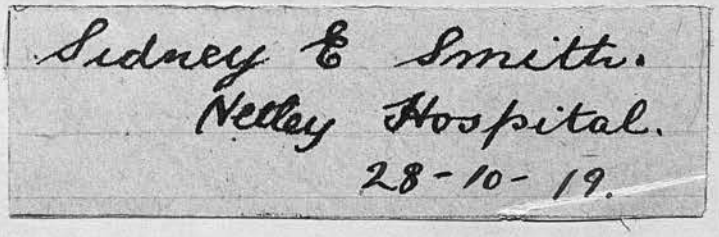
Written July before illness.

C.O. Durham.

Blighty

Smith

2. Handwriting two months after cerebellar symptoms.


Sidney E Smith.

Netley Hospital.

28-10-19.

he overshoots the patella by several inches.

2. Asynergia. Present to a slight degree in both upper limbs; absent in trunk.

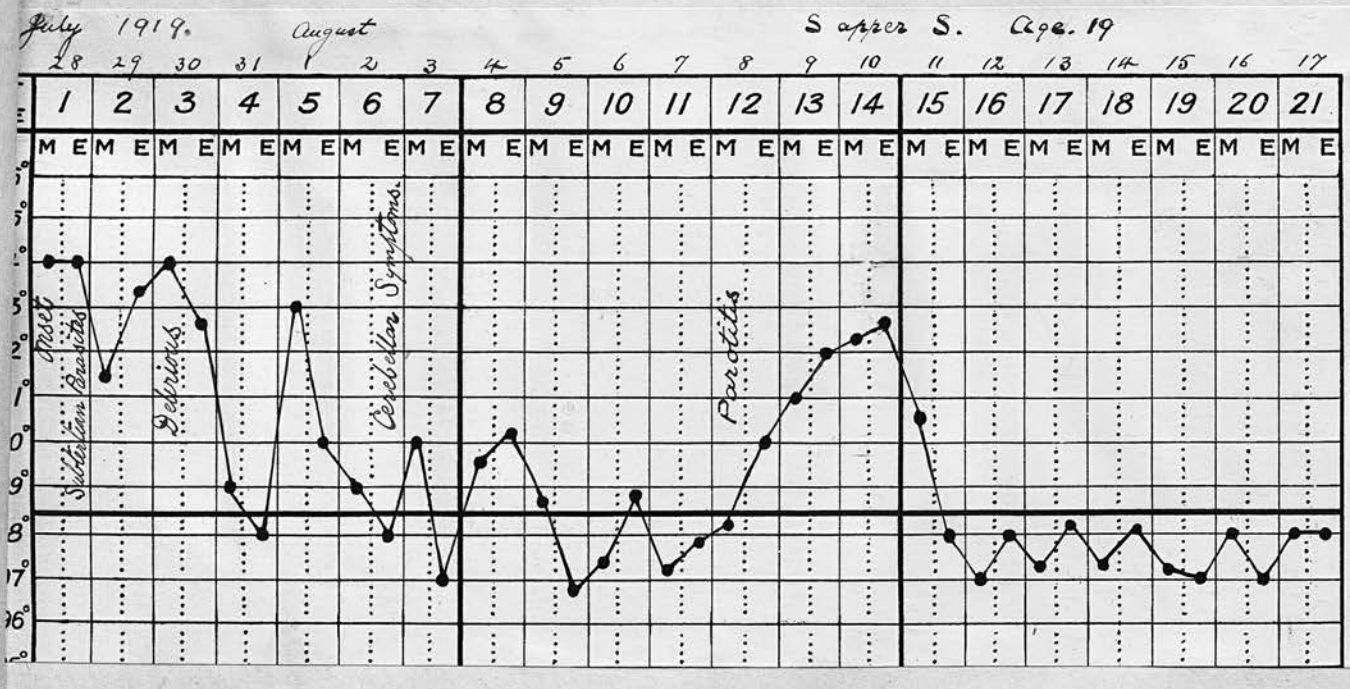
3. Dysdiadochokinesis. He has difficulty in making rapid alternate movements of pronation or supination with either forearm, but especially with left. Similarly, he has difficulty in rapidly opening or shutting palms of hands.

There is no cerebellar catelepsy.

Reflexes. Abdominals brisk and equal; plantars: flexor response both sides.

Tendon reflexes brisk and equal on the two sides.

Slight ankle clonus. Organic reflexes normal.



Comment.

In two of the cases recorded above (cases 1 & 4) the initial malarial attack was of great severity, and the subsequent cerebellar symptoms correspondingly severe, while in the other two cases the malarial paroxysm and the nervous complications were not of undue severity, and rapid improvement was noted. Each of the four cases presented some or all of the typical signs of a cerebellar syndrome, namely, dysmetria, asynergia, dysdiadochokinesis, and cerebellar gait, and in none was there involvement of the auditory apparatus. Defect of articulation was also found, and was one of the most marked symptoms.

Apart from one case reported by Lafora (6), the writer has not encountered similar examples in the neurological journals of the years 1914-1919, but the transient character of the symptoms in two of the cases suggests that this syndrome in malaria may escape recognition unless looked for, and may therefore be more common than a perusal of the literature would lead one to suspect.

The symptoms presented by these patients - and especially case 1 - are in many respects similar to the remote effects observed in a case of heat-stroke, which was fully reported by the writer.(7) From a consideration of certain experimental observations, it was concluded that in that case the cerebellar cortex, and in particular the Purkinje cells, had sustained a varying degree of

damage, and so given rise to the syndrome.
In cerebral malaria the pathological changes are apparently subject to considerable variation, and great stress is usually laid on the mechanical blockage of capillaries by parasites or blood pigment. There are reasonable grounds for believing, however, that a meningitis or encephalitis of malarial origin may frequently occur, and the view may be entertained that a meningo-encephalitis damaging the cortical elements of the cerebellum may have been the pathological basis in the above cases.

Hemiplegia and Monoplegia.

Malarial affections of the brain are occasionally met with which simulate cerebral haemorrhage. Although no such cases came under the writer's care, Castellani[8] mentions that he saw several in Macedonia, and they are also referred to by Paisseau.[9] In an interesting series of five cases recorded by Carmalt Jones[10], four were of the hemiplegic type, and in all with appropriate treatment there was a rapid recovery of motor power. Brosius[11] has described a typical example of the hemiplegic type. His patient was a girl aged 11, who was admitted to hospital in a state of semi-consciousness. The limbs on the right side showed

apparent absolute flaccid paralysis, but there was no difference in temperature between the limbs of the two sides ,and the face was not affected. A blood smear showed crescents and a few ring forms. Lumbar puncture yielded clear sterile fluid. On the second day following admission to hospital the hemiplegia disappeared, though the limbs affected were still weak.

Leri (12) has also given an excellent account of five cases of malarial hemiplegia occurring in young subjects in whom other possible causes could be excluded. He points out that there are two types of malarial hemiplegia: one,transient, which may be regarded as of toxi-infective origin, and a second which is permanent ; the latter he considers may be conditioned by emboli derived from the aorta .

Cranial Nerve Affections

Malarial complications affecting the cranial nerves appeared to be relatively infrequent, and the only cases treated in the Neurological department were examples of Facial paralysis. It must be remembered, however, that ocular affections would be sent to the Ophthalmic department, and those implicating the ear, nose, or throat, to the aural surgeon.

The following references to the literature may be made.

French observors (Paisseau[9]) have stated that complications affecting the sense organs occurred fairly often among Macedonian troops. Motor affections of the larynx were studied by De la Mothe, and Chaverne recorded cases of paralysis of the extrinsic and intrinsic ocular muscles.

Kirk,[13] who was brought into contact with a very large number of soldiers from the Salonica front, saw the following ophthalmic complications:-

1. Keratitis dendritica, due to a direct action of the malarial poison on the trophic ganglion cells of the fifth nerve.
2. External nerve paralysis. The 3rd., 4th., and 6th. nerves may be affected, more commonly the 6th.
3. A malarial neuralgia affecting the branches of the ophthalmic branch of the fifth cranial nerve.
4. Degenerative changes in the tissues of the optic nerve, producing a true malarial neuro-retinitis.

Blumenthal[14] has recorded a case of external rectus paralysis which occurred in a soldier, twelve days

after an attack of subtertian fever.

A case of malarial nystagmus, with no other manifestations in the nervous system is referred to in the Medical Supplement (15).

Optic neuritis in the course of cerebral malaria has been seen by da Matta (16). His patient was a boy aged 16; recovery occurred after six days treatment with quinine.

Dutheillet de Lamothe (17), working in the throat department of a Salonica hospital, observed 15 cases of laryngeal complications, which he classified as follows:-

1. Paresis or paralysis of the constrictors of the larynx and tensors of the vocal cords (11 cases).
2. Paralysis of the abductors (1 case).
3. Recurrent unilateral paralysis (2 cases).
4. A case in which the laryngeal paralysis was associated with an affection of the muscles of the soft palate, tongue, and face.

He favoured the hypothesis that such palsies are due to inhibitions of a toxic nature affecting the motor centres in the medulla oblongata.

An interesting case of multiple cranial nerve lesions is recorded by Sanz (18). A boy, aged 9 years, in convalescence from a severe attack of malaria, developed paralysis of half of the tongue, soft palate, and vocal cord on the left side. Four years later during another attack of malaria, he had a series of convulsions, followed by left hemiplegia.

Facial Paralysis.

The following examples of facial paralysis were all of the peripheral type.

Case 1.

Pte. F., aged 41, was admitted to the Neurological department on September 28, 1917. Prior to coming to Macedonia he had no illnesses of importance. Fifteen weeks previous to admission he was returning to duty after convalescence from a mild attack of malaria and diarrhoea, when he suddenly developed a severe attack of supraorbital neuralgia. He was in the train at the time, and his comrades called his attention to the asymmetry of his face. He was evacuated to a Base hospital, where he had a severe malarial attack. Fifteen days later he was transferred to the Neurological department

State on Admission.

General nuitrition good: no enlargement thyroid or spleen. Soft mitral systolic murmur at apex. Pulse 109 per minute.

Mental State: general intelligence poor; memory impaired, especially for recent events.

Neurological examination.

Speech and articulation normal. Pupils regular and equal: reactions normal. Visual fields normal; no nystagmus. Sense of smell and taste unimpaired. External ocular movements normal. Bell's paralysis on right side. Auditory acuity reduced (history of "gun deafness"). Tongue protrudes evenly. Sensory and motor functions normal.

Reflexes: Abdominals brisk and equal; plantars, flexor response. Knee and ankle jerks brisk and equal..

Face.

Asymmetrical at rest, and more so on movement. The lines on the right side of the forehead are almost obliterated; the right palpebral fissure is wider than the left. When patient attempts to close right eye, the eyeball rolls upwards and outwards: the right corneal reflex is abolished. If the observor's hand be brought abruptly towards the patient's face, he winks only with the left eye, but a contraction of the muscles on the right side of the mouth takes place - he "winks" with his mouth. Tapping the right supraorbital nerve causes a reflex contraction of the elevators and retractors of the right side of the mouth. Cestan's sign and Negri's sign negative.

When the patient looks down, the right upper eyelid relaxes, and the lids can be passively closed.

The patient cannot wrinkle brow nor frown on the right side: the right corner of the mouth droops. There is marked asymmetry on showing teeth, or on attempting to whistle. The right ala nasi does not move. When he protruded his tongue he has a noise "like the wind" in his left ear. He tends to bite his right cheek, and food collects between it and the teeth on that side.

Electrical reactions: Diminished response to faradism in all the muscles (innervated by the facial nerve) on the right side of the face.

Galvanic reactions normal.

On 11/10/17 the patient was transferred to another department, the facial paralysis having largely disappeared.

Case 2.

Pte. F. 9/K.O.R.L., aged 36, was admitted to the Neurological department on December 31, 1917.

Family history. Father paralysed: one brother has "rheumatic gout"; another brother has been invalided with "rheumatism".

Previous history. Syphilis when 17 years old. Rheumatism in hip joints when aged 26. Was in hospital in January 1917 with articular rheumatism affecting hip joints, and fingers of left hand. Patient had a primary attack of malaria on Sept. 12, 1917, and two relapses in November.

On the evening of November 4, a fortnight after his last attack, he experienced severe pain in the left side of the head and face. He woke next morning with a complete left-sided facial paralysis. There was no deafness or ear discharge. The pain remained for four days.

State on admission.

Patient is a well developed subject; there is an abcess scar behind the left angle of the lower jaw. Heart and lungs normal. Spleen palpable.

Mental functions normal.

Neurological examination.

No tremor: pupils normal. No nystagmus. All cranial nerves normal, except left seventh. No impairment

of taste or hearing.

Sensory and motor functions normal. Superficial and tendon reflexes normal.

Face.

There is complete paralysis of the left side of the face. The asymmetry at rest is increased on voluntary movement. There is neither emotional nor voluntary movement on the left side. The furrows of the forehead are absent, and the patient can neither wrinkle brow, nor frown on the left side. The eye is widely open on the affected side, and cannot be shut; tears run down the left cheek. When he attempts to shut the left eye, the eyeball rolls upwards and outwards, the cornea passing under cover of the upper lid. Cestan's sign positive. Negri's hyperkinetic sign positive. The left eyelid can be passively closed; if the lid be then passively raised, the globe moves from without inwards, so that the cornea which was previously directed upwards and outwards, is now directed upwards and inwards. The corneal reflex is abolished, and involuntary winking no longer occurs. The eye brims over with tears; the tip of the nose is drawn towards the sound side, the left naso-labial fold is flattened out, and the ala nasi shows no active movement.

The mouth is drawn towards the sound side: on the affected side its angle droops. When the patient smiles or shows upper teeth, the affected side alone moves. The patient cannot whistle, and art-

iculation is impaired. Food accumulates between the cheek and teeth. The left cheek flaps loosely during forcible expiration. Movement of the skin by the platysma does not occur on the left side.

Electrical reactions

Left facial nerve and muscles: no response to faradism. Galvanism: ACC=KCC

Progress.

11/1/18 Both patient's ankles are painful and swollen.

18/1/18 Wassermann reaction negative in blood and cerebrospinal fluid.

27/1/18 During ionisation patient is conscious of a metallic taste on the left side of the tongue.

13/2/18 There is distinct flattening of the muscles on the left side of the face.

15/2/18 The pseudo-deviation of the tongue to the right disappears when the left commissure of the mouth is pulled to the left. When the two sides of the tongue are examined with the faradic current, the muscles on the right side give an apparently brisker contraction than those on the left, but this is also due to the fact that movement of the left side of the tongue is impeded by the paralysed lips. If the mouth be held open on the left side there is no difference in the degree of contraction on the two sides.

27/2/18 Pleuritic friction is present on the left side of the chest in the neighbourhood of the nipple. There is evidence of very slight return of

conduction in the left facial nerve. On percussing the left supraorbital nerve, very slight retraction of angle of mouth occurs.

28/2/18 Evacuated by hospital ship.

Case 3.

Pte. P., 3/Cheshire Rgt., aged 38, was admitted to the neurological department on 14/2/18.

Family history negative.

Previous history. In childhood he suffered from diphtheria. His first attack of malaria was in July 1916. He had six subsequent relapses.

Present illness.

On January 1, 1918 he reported sick with headache, vomiting, and shivering. On the fourth day of his illness, his temperature having fallen to normal, he woke in the morning to find that the right side of his face was paralysed. There was no history of deafness or ear discharge.

State on admission.

He complains of his face being pulled to one side, of tears running down right cheek, and of blurred vision in the right eye.

Patient is thin and anaemic. Heart and lungs normal. Spleen enlarged and palpable.

Higher mental functions normal. No tremor. Speech normal; articulation normal. Pupils normal.

Cranial nerves normal, with the exception of the right facial nerve.No impairment of taste or hear-

ing.

Sensory and motor functions normal

Superficial and tendon reflexes normal.

Face.

There is incomplete paralysis of the right side of the face. The asymmetry at rest is exaggerated by voluntary movement. The furrows of the forehead are not quite so distinct as on the left side. The patient can feebly wrinkle brow and frown on that side. The right eye is slightly more open than the left and cannot be completely closed. Tears tend to run down the cheek. When patient attempts to close right eye, the upper lid moves slowly down for a distance of perhaps 2mm. No movement of the eyeball accompanies this effort. The right upper eyelid cannot be passively closed. Involuntary blinking on the right side does not occur, but the corneal reflex is not completely abolished. The right nasal orifice is narrowed, and shows no movement. The naso-labial fold is faint on the right side. The mouth is slightly drawn towards the normal side, and on the affected side its angle droops. When the patient smiles or attempts to show upper teeth, the affected side moves very slightly, The patient cannot whistle nor distend cheeks. Articulation is not impaired. Food accumulates between the cheek and teeth on the right side.

Electrical reactions.

There is diminished faradic excitability in the facial and muscles on the right side.

Galvanic reactions normal

Progress.

23/2/18 Slight return of motor power, especially in upper facial muscles.

20/5/18 Recovery is now practically complete.

Case 4.

Pte. H., A.S.C., aged 22, was admitted to the Neurological department on August 14, 1918.

Family history. Mother died of cancer: one sister of spinal disease.

Previous History

No history of rheumatism or ear disease. No serious illnesses in civil life.

His first attack of malaria was in July 1916: attacks of fever occurred every second day.

On August 7, 1918, five days after his last attack of malaria, he experienced a dull pain in front of the left ear; it gradually spread and by the third day was felt in the scalp, behind the ear, and in the lower jaw. There was no deafness or tinnitus.

On the evening of August 10 he noticed that he could not close completely the left eye, which "watered" freely. Next day he noticed that when eating liquids ran out of the corner of his mouth, and that food accumulated between his cheek and teeth on the left side. The left side of his face and scalp were very painful to pressure for several days, and the pain prevented him from sleeping at

night.

State on admission.

Patient complains of "stiffness" and loss of power in the left side of the face, and of pain in the left side of the head.

A thin healthy-looking subject. Heart and lungs nirmal; no enlargement thyroid or spleen.

Neurological examination.

Higher mental functions normal. Slight tremor of outstretched hands. Speech normal.

Articulation: labials and labio-dentals indistinctly pronounced.

Pupils slightly irregular in outline: normal reactions to light and on accomadation. Cranial nerves normal, except left facial. No nystagmua.

Sensory and motor functions normal.

Reflexes: Abdominals brisk and equal. Plantars: flexion both sides. Tendon reflexes brisk and equal.

Face.

There is almost complete paralysis of the left side of the face. The asymmetry at rest is exaggerated by voluntary movement. The only voluntary movement which the patient can execute on the left side is partial closure of the eye. The furrows of the forehead are absent on the left side, and he is unable to wrinkle the brow or to frown on that side. The left eye is more widely open than the right, and can only be partially closed; when he attempts to shut it the eyeball rolls upwards and to the left.

Case 4. Pte. H. Left facial paralysis

1. Face at rest.

2. Showing teeth.

3. Smiling.

It may, however, come to rest in the middle line. Cestan's sign negative: Negri's sign positive. Tears tend to run down the left cheek, and vision is blurred. Spontaneous winking occurs on the left side, but movement of the upper lid is of smaller amplitude than on the normal side. Reflex winking of the left eye does not occur. The left corneal reflex is absent. The tip of the nose is drawn slightly towards the sound side, the left naso-labial fold is flattened, and the left angle of the mouth is lower than the right. When patient smiles or shows teeth the healthy side alone moves. The patient cannot whistle and articualtion is impaired. Food accumulates between the left cheek and the teeth. He cannot distend cheeks tightly - air escapes through the left side of the mouth; the left cheek flaps loosely during forced expiration. The left platysma does not contract. There is no disturbance of cutaneous or deep sensibility, but the left facial nerve is definitely painful to pressure either in front of mastoid process or on mandible. Percussion of the supraorbital nerves evokes contraction of the corrugator supercilii to an equal degree on both sides.

Electrical reactions.

Faradism: Marked diminution of excitability in muscles supplied by superior division, diminution in muscles supplied by middle division, normal reaction in muscles supplied by lower division of left facial nerve. Galvanic reactions normal.

Taste is normal on both sides.

Progress.

18/8/18 The diminished response to faradism is more marked. He sweats to an equal extent on both sides of face.

21/8/18 Both tympanic membranes are normal. He complains of tenderness on pressure along left ramus of lower jaw.

22/8/18 Slight improvement. He can now almost completely close his left eye. Percussion of left facial nerve causes very slight contraction of left labial muscles

26/8/18 Considerable return of motor power in left face.

1/9/18 Weakness is now only noticable in lower facial muscles

5/9/18 Discharged recovered.

Case 5.

Cpl. G., A.O.C., aged 27, was admitted to the Neurological department on September 18, 1918.

Family history: one sister died of diphtheria.

Previous history

Scarlet fever and influenza in childhood. Enlarged cervical glands four years ago. Has had a facial tic for years.

His first attack of malaria was in August 1917, and he has had a number of slight relapses since. The last attack of fever occurred five days ago.

Present illness.

On September 5 patient noticed on getting up in

the morning "stiffness" of the right eye: it seemed heavy, having an inclination of its own to close. He felt nothing wrong with the left side of the face. Twenty four hours later he noticed "stiffness" of the muscles of the left side of his mouth, which was most obvious when he was eating or drinking. He could not masticate properly, and fluid escaped from the left corner of his mouth. When washing he had difficulty in preventing soap entering his left eye. The condition gradually became worse, and on September 10 the paralysis was complete. During the onset of these symptoms he had no earache, headache, or fever, although he did not feel well.

State on admission.

Complaint: lack of control over left side of face and feeling of heaviness in right eye.

A thin anaemic man: wears convex glasses. Pulse 80: apical systolic murmur. No enlargement thyroid or spleen.

Neurological examination.

Higher mental functions normal. Speech normal. Articulation: words beginning eith the letter "B" are indistinctly pronounced. Pupils normal. Cranial nerves normal, except left facial. No nystagmus. No abnormality detected in right eye. Sensory and motor functions normal. Reflexes normal.

Face.

The face is slightly asymmetrical, the lines on

the left side being less distinct than those on the right. The patient cannot raise left eyebrow as high as the right, nor can he frown so well on the left side. The left palpebral aperture is wider than the right, and he cannot close left eye completely: the eyeball moves up very slightly when he attempts to do this. Spontaneous and reflex winking is preserved. He cannot whistle, and only exposes teeth to a slight extent on the left side. Platysma contracts well. When he inflates cheeks, air escapes on the right side of his mouth. No deafness, and no tenderness over facial nerve.

Taste normal.

Electrical reactions.

Slight diminution in faradic excitability of left facial muscles. Galvanic reactions normal.

Progress.

20/9/18. Attack of malaria to day.

21/9/18. Paresis of face is rapidly disappearing.

25/9/18 Facial paresis has disappeared.

Case 6.

Pte. S.. R.A.MC., aged 25, was admitted to the Neurological department on October 7, 1918.

Family history.

Father died of heart disease; one sister died of tuberculosis; another sister insane.

Previous history.

Has always been delicate. "Abscess of spine" when 8 years old. His first attack of malaria was in

August 1918; two relapses.

Present illness.

On September 30, two days after an attack of malaria he was suddenly seized with severe shooting pains in the left temple and left side of the neck. There was no earache or deafness. After twenty four hours the pains disappeared, leaving facial paralysis. He noticed that he could not close left eye, nor smile on the left side of the face.

State on admission.

Complaint: "stiffness" of the left side of the face.

A weak degenerate subject. Enlarged cervical glands on right side. No enlargement of spleen.

Neurological examination.

General intelligence poor; coarse irregular tremor of limbs, increased by emotional excitement.

Speech normal; articulation indistinct. Pupils dilated. Cranial nerves normal, except left facial.

Sensory and motor functions normal. Superficial and tendon reflexes brisk and equal.

Slight exophthalmos and Von Graefe's sign positive.

Face.

There is complete paralysis of the left side of the face; there is neither voluntary nor emotional movement. Very slight reflex winking of the left eye may be observed. He cannot frown nor wrinkle brow on the left side, the normal furrows being absent. The left eye is more widely open than the right, showing sclera above and below cornea.

When patient attempts to close the left eye, the

eyeball rolls upwards under cover of the upper lid, but only deviated outwards to a very slight degree. Cestan's sign negative; Negri's sign negative.

Tears run down the left cheek. Spontaneous winking does not occur on the leftside, and the left corneal reflex is absent. The tip of the nose deviates to the right, and the left ala nasi does not move. The mouth is markedly assymetrical, being drawn over to the right. When patient smiles or attempts to show teeth, the healthy side alone moves. He cannot whistle and articulation is impaired. He is unable to distend cheeks, and food collects between the cheek and teeth on the left side.

Auditory acuity and taste are unimpaired. The left facial nerve is not apinful to pressure.

Electrical reactions.

There is no response to faradism on the left side. The left facial muscles are hyperexcitable to galvanism. KCC>ACC.

Progress.

The patient passed out of observation before any improvement had occurred.

Illustrations. Case 6. Left facial paralysis.

1. Appearance of face at rest.
2. Patient attempting to expose teeth.

1.

2.

Comment.

Since the facial nerve is paralysed more frequently than any other nerve in the body, it is open to doubt whether malaria was causally related to the cases described above.

In Case 1. chill must be reckoned with as a factor in the production of the paralysis, as it appeared when the patient was returning to his unit in a railway train, a circumstance which doubtless exposed him to a draught. In his case the malarial infection may have acted as a predisposing cause.

Case 2. was of interest for the paralysis was profound. Moreover, the patient was of the rheumatic diathesis, and gave a history of having contracted syphilis in youth. In the neighbourhood of the point of emergence of the paralysed facial nerve there was an old operation scar which by incarceration of the nerve trunk may have formed a locus minoris resistantiae. It is evident, then, that malaria was only one of several possible etiological factors, between which it is difficult to discriminate.

In the remaining four cases the paralysis occurred a few days subsequent to malarial attacks, and was not related to any known other etiological factors.

The severe pain which was complained of by one patient (case 4) might be attributed to a simultaneous (neuritic?) affection of the sensory nerves.

The rapid recovery in several of the cases is worthy of note, and in case 2, which responded very slowly to treatment, the earliest sign of recovery was a reflex contraction of certain muscles, elicited by percussion of the supra-orbital nerve.

This sign is of undoubted prognostic value, for it precedes the return of voluntary power in the paralysed muscles.

Spinal Cord Affections.

Malarial affections of the spinal cord must be regarded as rare. Two cases came under observation in the Neurological Department, and both were complicated by the co-existence of a polyneuritis. Castellani (8) states that he has seen one case of clinically typical transverse myelitis, and Sabatucci (26) has recorded two cases of complete paraplegia in soldiers who contracted malaria in Macedonia.

Case 1.

Pte. L., 1/Suffolk Rgt., aged 21, was admitted to the Neurological Department on Sept.9, 1917.

Previous history. It was impossible to obtain data with regard to his family history and previous health. The following notes were made by his Regimental medical officer:- "Had fever on Aug. 27th. when his temperature was up for one evening only. Pupils: left is slightly larger than the right; both react normally. No anaesthesia for touch, pain, or temperature. Knee jerks absent; plantar reflexes flexor. Slight tremor tongue, and incoordination of arms. Can only stand with difficulty. Rombergism present. (Sept. 5, 1917).

On Sept.8 his condition was as follows:-

Pupils and cranial nerves normal; no cutaneous anaesthesia or analgesia. Hand grasps feeble: upper limbs slightly feeble; no movements impossible. Cannot sit up without using hands; umbilicus pulled up in attempt to sit up; abdominal muscles feeble Cannot lift either lower limb off bed; all movement of lower limbs feeble, but none impossible. No muscular tenderness. Knee and ankle jerks absent. Plantars feebly extensor. Abdominal reflexes: lower absent, upper brisk. Pulse rate rapid, 106 to 124. No fever: respirations normal.

State on admission.

He complains of inability to use lower limbs or to sit up, of weakness in both upper extremities, and of tingling in finger tips. The illness commenced on 27.8.17. with fever (about 101.2 F.). As far as

can be gathered the paresis appeared very suddenly.

Neurological examination.

Pupils normal. All movements of face possible but weak; he cannot close eyes firmly, show teeth, nor whistle. There is paresis of the palate, and the tongue cannot be protruded fully. Mucus accumulates at the back of the throat owing to difficulty in swallowing. Articulation is nasal, indistinct, and not easy to understand. The other cranial nerves are normal.

Sensory functions. Tactual hypoaesthesia of fingers and hands, fading off towards wrists, where normal sensibility is preserved. On the lower limbs there is blunting to touch from about the middle of the thighs downwards. Pain sensibility is normal, but on the lower limbs the stimuli evoke movements of rhythmic alternate progression like those of "narcosis progression". Pressure pain on fingers and hands gives a sensation of greater radiation than elsewhere. Pressure on the crural and peroneal nerves is painful; there is no muscular hyperaesthesia. Vibration sense is diminished on the finger tips, and on the peripheral parts of the legs. There is no disturbance of temperature sense; Compass test: the threshold is raised to two simultaneously applied stimuli on the legs. Joint sense: The recognition of passive movement is impaired in the interphalangeal, the knee, and the ankle joints Motor functions. Weakness in all muscles of both upper extremities, and more pronounced in the distal segments. The movements executed by the intrin-

sic muscles of the hands are especially weak. Extensor movements are weaker than flexor. He is unable to sit up even with the aid of his arms, and when he tries to do so the umbilicus is pulled up. All movements of the lower limbs are feebly performed.

Reflexes. Plantar reflexes, flexion. All other reflexes, superficial and deep are absent. Organic reflexes normal.

Progress. 10.9.17. Slept badly last night. His respirations are shallow and thoracic in type. Bowels moved well after enema.

11.9.17. Much better today, and movements of upper limbs stronger. Muscles of thighs and legs flaccid. Plantar reflexes now absent. When told to move his toes he performs rhythmic alternate movements of progression comparable with those of a cat in fairly deep narcosis.

Lumbar puncture performed. Cerebrospinal fluid: normal tension: of a greenish opalescent colour; 12 lymphocytes per c.mm. Globulin content increased: sterile. Blood film shows one benign tertian ring.

12.9.17. Worse. Last night breathing very difficult Temperature rose to 102 F. in early morning; he vomited at 11 a.m. Respirations 48 per min. Pulse 130. Cyanosed. At 2 p.m. the movements of his limbs were much weaker. When told to move his toes the rhythmic progression movements still occurred. He is almost inarticulate. At 4.30 p.m. he became very cyanosed his breathing being very laboured and

shallow. He appeared stuporose, but woke up if spoken to. At 5 p.m. a swab was taken from his throat. No Klebs-Loffler bacilli found.

He gradually sank and died at 7.30 p.m. of respiratory failure.

Post Mortem examination. 13.9.17. 9.30 a.m.

Rigor mortis marked. Post mortem lividity present. Lungs healthy. Heart not examined. Bowel distended eith gas. Spleen enlarged. Kidneys congested.

Central nervous system. Meninges and sinuses normal Brain large: no cortical atrophy. Congestion of grey matter and oedema of white. No excess of c.s.f. No granulations. Slight adhesions between temporal poles and inferior frontal regions. Medulla oblongata, no naked eye change.

Spinal cord. Membranes normal. The sixth thoracic segment and the upper half of the seventh has a swollen appearance, and a distinctly larger circumference than the segments above and below it. On the left side of the posterior columns in the 7th. T. segment a small haemorrhage can be seen; the cord substance at this level is of the consistence of cream cheese.

Report on sections from Base Laboratory 1.10.17.

Right phrenic nerve: scattered Marchi degeneration. Right musculo-spiral nerve: advanced Marchi degeneration. Cervical region cord: Marchi degeneration all through.

Cervical Cord: Haemorrhages into cord in areas which are necrosed. Vessels congested: no thrombosis seen. Extreme degree of cell degeneration - all

stages of degeneration.

Dorsal Cord: Congestion of vessels in nerve root bundles, and thrombosis in meninges. The nerve cells are shrunken and show all stages of degeneration.

Lumbar Cord: Nerve cells show all stages of degeneration, many being much distended and their nuclei extruded.

Pte. L. aged. 21. Myelitis

	AM			PM			AM			PM			AM			PM			AM			PM		
Time	2	6	10	2	6	10	2	6	10	2	6	10	2	6	10	2	6	10	2	6	10	2	6	10
Bowels																								
Urine																								

Temperature (Fahrenheit)

107° 106° 105° 104° 103° 102° 101° 100° 99° 98° 97°

Normal Temperat of body.

Blood 3.1. Ring

Urine 22 ×× Albumen

Died 7.30 P.M.

Day of Dis.	5	6	7	8
Pulse.	116	120	116 122	100 108 136
Resp.				48 48
Date.	September 9	10	11	12

Entered at Stationers Hall. Printed and Published by Wodderspoon & Co. 6. Gate.

Case 2.

Pte H., A.C.C., aged 23, was admitted to the Neurological department on November 16, 1917.

Family history negative.

Previous illnesses. Jaundice in early childhood. Diphtheritic paralysis when 10 years of age; neck, arms and legs affected; has had choreiform movements affecting face and limbs ever since.

Came to Salonica on Jan. 1 1017: no illnesses, except diarrhoea in April.

Present illness.

His illness commenced on November 2 with a feeling of malaise, headache and giddiness; he had a temperature of 104 F. He improved under quinine treatment and by November 9 was able to get about. Two days later he had a return of headache, and on November 12 he felt his lower limbs weak, and fell. His pulse was somewhat rapid.

On November 14 he complained of pain in the back, and was unable to walk. His plantar reflexes, his knee and ankle jerks were found to be absent. All voluntary movements of limbs appeared weak. His pharynx was insensitive to touch. On November 16 he felt tingling and numbness in his hands and feet.

State on admission. 18.11.17.

Patient complains of numbness in hands and feet, of general weakness, and of difficulty in making water.

Neurological examination.

Neurological examination.

Higher mental functions normal. Speech normal. articulation slightly indistinct and nasal. Involuntary movements of the extensors of the toes can be observed, occurring irregularly every few seconds.

Pupils dilated and equal: reactions normal. No nystagmus. Lower jaw deviates very slightly to the right when he opens mouth. Palate moves feebly on phonation. Tongue protrudes straight.

Sensory functions. To cotton wool touches there is slight blunting on fingers, dorsal surfaces of feet and toes. To pinprick there is blunting on the ulnar 1½ fingers of the right hand. There is analgesia of the lower limbs (except soles) up to the lower border of the patellae. The ulnar, the external popliteal nerves, the calves and the soles of the feet are hyperaesthetic to pressure. Joint sense impaired in fingers, and markedly impaired in all joints of lower extremities. Vibration sense lost in lower limbs.

Motor functions.

Diffuse paresis of all muscles of upper extremities. Patient cannot raise either upper limb above the horizontal (yesterday he could scratch head with either hand). The fingers are feebly abducted, and no adduction of right minimus is possible. The right minimus and annularis are in a position of "claw hand". No individual movement of the left upper limb is impossible. He is unable to sit, and

the movements of diaphragm and intercostal muscles are shallow. His cough is feeble.

Lower limbs: he cannot raise right limb off the bed, and left heel for only a few inches. The movements at toes and ankle joints are fairly good.

The hamstrings and quadriceps are feeble. He is only able to draw up his knees through a very limited range. All the muscles are hypotonic.

Reflexes: Abdominal and plantar reflesex absent. Arm jerks diminished right side, absent left. He has difficulty in making water, and his bowels have not moved for three days.

Cerebrospinal fluid: pressure increased; marked excess of globulins.

Progress.

18/11/17 6p.m. Articulation distinctly nasal. Movements of facial muscles normal, except that he cannot whistle. No sensory disturbance on face; corneal reflexes present. There is anaesthesia of pharynx, analgesia of right side of palate, and absent palatal and pharyngeal reflexes.

Plantar reflexes now give a flexor response, and when being elicited there is synchronous and very marked extension of opposite limb.(right or left).

19/11/17.

Is said to have been delirious in early part of last night. Mental state now normal. He complains of headache, of weakness of voice, and of pain in the small of the back. Slight oedema of face this morning. Articulation more indistinct. Pupils

contracted. On opening mouth lower jaw deviates to right, and left condylar process becomes prominent. Left masseter less powerful than right. Diminution of salivary secretion.

There is analgesia to pinprick on tongue, mucus surface lower and right half upper lip, hard palate, and inner side of cheeks and pharynx.

paresis of facial muscles, especially left. He cannot whistle nor forcibly close eyes.

To cotton wool touches anaesthesia of right palm up to fold of wrist, and 1½ ulnar fingers on dorsum. On left hand anaesthesia up to 3 fingers breadth above wrist. To pinprick blunting on fingers (palmar surfaces) and on dorsal surfaces over middle and terminal phalanges of minimus and annularis.

Joint sense is impaired in both upper limbs, and is greater in distal joints. Vibration sense is lost in the distal two phalanges of annularis and minimus of right hand.

Lower limbs. Slight blunting to cotton wool touches on toes; no analgesia. Joint sense now impaired in knees, especially left. All muscles of the lower limbs are tender on pressure.

Motor functions.

Slight wasting of cervical muscles. He is just able to raise either arm up to the level of his mouth, but no further. Diffuse weakness both upper limbs, most marked in shoulder muscles and intrinsic muscles of hands. "Claw" hand is more marked in the right hand. Diaphragm feeble. Umbilicus

moves very slightly up when patient attempts to flex neck. Intercostals weak.

Lower limbs. Diffuse weakness, more marked on right side. Toe movements good. Marked weakness dorsi-flexors of ankles, quadriceps, and hamstrings.

Reflexes. Supinator and biceps jerks absent. Knee and ankle jerks absent; abdominal reflexes absent.

Plantars: right, absent: left extensor response.

6 p.m. Seen by Consulting Physician who regards case as one of profound polyneuritis.

20/11/17

Complains of very severe pain in "small of Back", and of more weakness in upper limbs. No difficulty in micturition. Abrupt, involuntary choreiform movements can be observed affection face, left shoulder girdle, and lower limbs.

Slight paresis left facial muscles: when asleep left eye is habitually a little open.

Joint sensibility is impaired in all four limbs, and sense of posture is grossly defective - when one leg is crossed over the other he describes them as being "about a foot apart".

6 p.m.

Weakness of upper limbs more marked; has especial difficulty in opening and closing left hand.

He complains of a constant desire to spit, and has great difficulty in expelling fluid from the mouth. Deglutition is difficult.

Laboratory report: No Klebs-Loffler bacilli from throat.

21/11/17

Late last night respirations became irregular and embarrassed; promptly relieved by administration of oxygen.

Patient is constantly expelling frothy sputum from his mouth. Voice markedly hoarse. Expiratory stridor. Tendancy to Cheyne-Stokes breathing; expiratory excursions of thorax vary much in rhythm and amplitude: stridor increases after feeding.

Urine, blood, and cerebrospinal fluid normal as regards pathogenic organisms.

6 p.m

Breathing is much embarrassed; secretion collects in pharynx and larynx and patient cannot expel it. He states that he is choking. Laryngotomy considered, but crisis passed off after administration of atropin.

11 p.m. Breathing again greatly distressed.

22/11/17

Temp. 98.4 F. Pulse 140: small and irregular. Respirations shallow, feeble, and 50 per minute. Very slight cyanosis. Mind quite clear.

6 p.m. Appears weaker: pulse 120 per minute, slightly irregular and small.

23/11/17

Respirations noisy, and secretion again collecting in throat. Mind quite clear . Temperature 100.4 F., respirations 52, pulse 150.

He died suddenly at 6.30 p.m., having remarked several minutes before that he would be "all right".

Post-Mortem Examination.

An autopsy was held 19 hours after death.

Body that of a male adult subject ; pupils unequal. Rigor mortis marked in limbs. Post-mortem lividity marked on face and posterior aspect trunk.

Attitude of limbs: slight "claw foot" both sides.

Brain Antemortem thrombosis in Sup. long. sinus. Dura mater normal; a few subpial haemorrhagic extravasations in parieto-occipital areas. Slight temporal pial adhesions. No atrophy. Grey matter normal in appearance. Marked oedema of whole brain; no ventricular dilatation. Basal ganglia normal.

Congestion of grey matter of pons and medulla.

Spinal Cord.

Venous congestion of pia-arachnoid. At the 8th. thoracic segment the investing pia has a rusty appearance, and there is a definite complete softening of the cord, about 7.5 m.m. in length. The right vagus nerve has a reddish hue and looks slightly swollen.

Heart: slight hypertrophy of left ventricle, and dilatation of right chambers, which are filled with red and white thrombi.

Lungs: No pleural adhesions: hypostatic congestion right lower lobe. Liver slightly enlarged. Spleen also a litle enlarged and semidiffluent. Kidneys congested: otherwise normal.

Comment.

The fatal termination in both the cases recorded above justifies the conclusion that, apart from instances of cerebral malaria, complications affecting the spinal cord must be regarded with considerable anxiety.

A certain number of points of resemblance were found in these two cases. In both there was implication of the cranial and peripheral nerves to an extent which gave a clinical picture more suggestive of a profound polyneuritis than of acute myelomalacia. Neither case gave a history of numerous malarial attacks, and the febrile periods which preceded the onset of the nervous symptoms could hardly be regarded as typical of malaria. It is true that in one instance blood examination gave positive results, but no mention of the presence of parasites in tissue removed after death, was received from the Base Laboratory. It is therefore a point of some importance to exclude other possible causes.

The disease which in its clinical features most closely resembles those recorded above is that known as Acute Infective Polyneuritis. Its manifestations have been the subject of an exhaustive study by Bradford, Bashford, and Wilson.(19)

It is a diffuse affection of the nervous system, the palsy being of sudden onset, widespread, and peculiar in its distribution - the proximal segments of the limbs being especially implicated.

Sensory disturbances occur at the periphery of the limbs, and the tendon reflexes are usually lost. Somewhat similar symptoms were present in both cases of malarial myelomalacia, but here the resemblance ends, for in contrast with the findings in febrile polyneuritis, the cerebrospinal fluid was in each case definitely abnormal, and gross softenings of the spinal cord were found after death. In its morbid anatomy acute febrile (or infective) polyneuritis resembles acute poliomyelitis rather than myelomalacia.

The possibility that both cases were examples of either poliomyelitis or Landry's paralysis can hardly be entertained inview of the marked disturbances of sensation which were found.

In certain parts of the world - especially Australia-very similar symptoms have been observed in cases of human infestation by ticks (20) (Ixodes holocyclus). In a country like Macedonia where there are dense areas of scrub it is probable that more than one form of pathogenic Ixodidae may exist, although the tick of relapsing fever appeared to be the only known variety.

Certain investigations undertaken by T. Graham Brown and the writer on cases of spinal injury, (21) led to the conclusion that the presence of rhythmic phenomena in man is of diagnostic value. It was mentioned in the clinical notes in Case 1. that the prick of a pin on the sole of either foot started rhythmic alternate movements of flexion

and extension at ankle, knee, and hip of both lower limbs. On the evidence of this phenomenon a diagnosis was made of myelitis in the lower thoracic part of the spinal cord. The lesion was found to be actually in the sixth thoracic segment.

Polyneuritis.

A malarial polyneuritis has long been recognised, and is said to be the commonest nervous complication. Cajazeira[22], however, denies the existence of malarial polyneuritis, and attributes its presence in malarial subjects to alcoholic intoxication, or to improper feeding, which gives rise to beri-beri. That it is a definite clinical entity is clearly shown by the following examples.

Case 1.

Pte. R., Royal Serbian Army, aged 23, was admitted to hospital on June 5, 1917.

History.

Five days prior to admission patient had a typical malarial paroxysm, which was followed by weakness of the upper limbs. He was suspected of malingering, and therefore admitted to hospital for observation.

State on admission.

Slight fever: rapid pulse and constipation.

Complaint: severe pains in both upper limbs.

There is anaesthesia along the ulnar aspect of left upper limb, and definite weakness of biceps, triceps, and forearm muscles; he cannot extend wrists.

Reflexes: arm jerjs absent. Knee jerks: slight contraction of quadriceps, but no movement of legs.

18/6/17. Vomited last night; pulse rapid.

Neurological examination:

Patient complains of pains in the head and limbs.
Fibrillary tremor can be seen in the muscles of the left shoulder girdle.
Pupils and cranial nerves normal; no nystagmus.
Sensory functions.
To cottonwool touches there is blunting over the whole of the left upper limb, and on both aspects of left shoulder. To pinprick there is an area of diminished sensibility on both aspects of left shoulder; there is no cutaneous sensory disturbance in the lower limbs.
Vibration sense diminished in the left hand.
Joint sense normal.
Pressure on certain nerve trunks is very painful, especially left ulnar, left brachial cords, right and left sciatic nerves; the muscles of the left shoulder girdle are very sensitive to pressure.
Motor functions:
Patient cannot elevate either upper limb above the horizontal, abduction and adduction are feeble, and backward movement of the left upper limb is hardly possible. The forearm and hand muscles are diffusely weak on both sides. There is weakness of the intercostal muscles, and those of both lower limbs.
There is obvious wasting of the muscles of the shoulder girdles and upper arms and especially on the left side.
Reflexes: right abdominal reflexes absent: left present; plantar reflexes absent.

Right supinator jerk present: other arm jerks absent. Right knee jerk present, but much diminished. Left absent; sphincters normal.

Electrical reactions:

Faradism of the right brachial plexus produces very little muscular contraction, and only in the long flexors and extensors of digits. No response to faradism of median and ulnar nerves at wrist. The supinator longus contracts very feebly.

Left side: faradism of brachial plexus causes feeble contraction of flexors of digits; no other response: no contraction of supinator longus.

26/6/17.

Vomits every day after food; pulse 100 per min.

Higher mental functions normal.

There is marked fibrillary tremor of muscles of shoulder girdles and upper limbs, especially on the left side.

Speech and articulation normal; pupils and cranial nerves normal.

Sensory functions.

No cutaneous anaesthesia or analgesia. Vibration sense diminished on nails and bony prominences of left upper limb.

All accessible nerve trunks are very sensitive to pressure, especially those on the left side.

Motor functions.

Patient cannot abduct left upper limb to the horizontal, nor can he maintain its posture when it is placed in a vertical position above his head. He can perform these actions on the right side.

The left latissimus dorsi does not contract when the patient coughs.

Hand grasps: right, fair; left, very weak.

Both lower limbs show diffuse weakness, equal on the two sides.

There is marked wasting of the shoulder girdle and upper arm muscles, especially on the left side.

Reflexes: arm jerks absent. Knee jerks just present.

Right ankle jerk diminished: left normal.

The patient was evacuated to the Base on June 27.

Case 2.

L/Cpl. W., M.G.C., aged 22, was admitted to the Neurological department on July 21, 1918.

Family history.

Mother died of "diseased nerves" of 21 years duration. One uncle paralysed.

Previous illness.

None in civil life except measles in childhood.

His first attack of malaria was in June 1917, and he has had 15 subsequent attacks. The last attack which occurred on 13/7/18 was the worst he has had. It was ushered in by headache, shivering, vomiting and fever (temp. 103.6), and on the second day he experienced pins and needles in the soles of his feet; his legs felt weak and heavy when he attempted to walk. On 17/7/18 pins and needles was felt in the finger tips, and on 18/7/18 he was hardly able to walk.

His state on admission to a general hospital on 21/7/18 was as follows:-

No diplopia; ocular axes parallel. No asymmetry of face, but paresis of all left facial muscles. Patient cannot close completely the left eye, and in attempting to do so the left eyeball rolls upwards and outwards. When retracting the angles of the mouth, the left naso-labial fold is much less marked than its fellow. The left eye lachrymates freely, and winks less frequently than the right. He has considerable difficulty in whistling.
The pterygoid and masseter muscles contract powerfully, but are painful to pressure. The palate moves normally, and the tongue protrudes evenly. He has difficulty in swallowing.
Sensory functions.
To light cotton wool touches there is complete loss in glove distribution, and blunting which fades away at the elbows. On the lower limbs there is complete anaesthesia up to the knees.
To pinpricks there is complete loss of pain sensibility on the dorsal surfaces of the hands. There is hyperaesthesia to pinpricks on the palmar aspects of the hands, and on the lower limbs as high as the knees; on the hyperaesthetic areas the sensation evoked by a pinprick lasts for 4 seconds.
Extreme cold (ethyl chloride spray) is described as a "bit cold" on the dorsum of the hands: normal on remainder of upperllimbs. On the lower limbs below the knees it is described as unpleasantly hot: normal on the thighs.
Heat (test tube at 80 C.) is appreciated as such on the palms,but is not unpleasantly hot, as it

is on normal areas; on the lower limbs it evokes painful sensations of heat on the soles and thighs, but lacks the pain element on the legs.

Two-point threshold: no threshold on dorsal surfaces of hands or on legs; localisation defective on toes.

Vibration sense lost in phalanges and metacarpal bones: also in bones of lower limbs.

Joint sense: normal upper limbs; recognition of passive movement and its direction impaired in toes.

The following muscles are tender to pressure:- Masseters, deltoids, and all other muscles of upper and lower limbs. Pressure on either ext. popliteal nerve or on tendo achillis elicits pain. The ulnar nerves are relatively insensitive to pressure.

Motor functions.

Diffuse motor weakness in upper limbs: no individual movements impossible, except approximation of minimus to pollex, either side. Fine movements of fingers clumsily performed, and abduction feeble.

Diaphragm intercostals, and abdominal muscles normal. Patient cannot maintain sitting posture in bed unless he supports himself with his arms.

Lower limbs: marked paresis but no individual movement impossible. He can only elevate either limb for a few inches off the bed, and while doing so there is marked tremor. Extension of lower limbs against resistance is very weak.

All limb muscles feel flaccid and toneless; there is slight wasting of peronei.

Reflexes: Corneal and palatal reflexes present.

Epigastric and abdominal reflexes present, but diminished. Plantars: no toe movement, but contraction of hamstrings and tensor fascia femoris muscles.

All deep reflexes abolished. Organic reflexes normal.

Electrical reactions.

Faradism: upper limbs normal. Lower limbs, diminished excitability, especially in peronei and calf m muscles.

Galvanism: KCC>ACC all muscles.

Cerebrospinal fluid.

Pressure increased; faint yellow tint. Sugar present; globulins in excess. Cell count normal.

There is hyperidrosis of the fingers and distal halves of the feet.

Progress.

2/8/18. Deglutition now normal, and left sided facial paresis has almost disappeared- no noticable difference in closure of eyes. There is diminution of faradic excitabilty in the facial muscles of both sides, but especially right. Lumbar muscles, glutei and hamstrings contract sluggishly: upper limb muscles contract briskly to faradism.

The finer movements of the hands are lost - he cannot button his shirt. There is a slight degree of astereognosis, and he cannot distinguish textures.

3/8/18 Slight fever this evening. Blood film negative. There is a moderate degree of wasting of first dorsal interossei.

Case 2. L/Cpl. W. Malarial Polyneuritis.
Illustration to show wasting of interossei;
The atrophy of the first dorsal interosseus
muscle is very apparent in the left hand.

10/8/18. Complains of pains in lower limbs at night. He is now able to oppose thumb and little finger.

13/8/18. Anaemic: pulse 120 per min. Apical systolic murmur, not conducted. Spleen palpable. Cranial nerves now normal, and less incoordination of upper limbs.

Sensory functions: there is hyperaesthesia to pin prick on palmar surfaces of hands and fingers, and in sock distribution; the forearm muscles are slightly tender on pressure, and those of the calves markedly so. Other forms of sensation normal.

Motor functions: weakness of intrinsic muscles of hands and of extensors of wrists. Lower limbs are diffusely weak.

Gait: Patient is able to stand, and to walk with feeble short steps: he exhibits ataxia.

There is diffuse wasting of the muscles of the upper limbs, and markedly of the dorsal interossei.

Reflexes: abdominals brisk and equal; plantars, flexion. Tendon reflexes absent, except left supinator jerk. Organic reflexes normal.

Electrical reactions: to faradism diminished response in intrinsic muscles hands, calf muscles, and peronei.

17/8/18 Shaved himself for the first time today; malarial paroxysm this afternoon.

19/8/18 Temperature normal. Benign tertian rings present in blood. Patient states that the palms of his hands are unnaturally sensitive to all forms of stimuli

26/8/18 Patient is now able to walk about the ward.

18/9/18 No disturbance of cutaneous sensibility. Muscles of lower limbs are slightly sensitive to pressure. There is still weakness of extensors of forearms, and of all muscles of lower limbs. Wasting of first dorsal interossei of hands. Gait normal. Superficial and deep reflexes normal, save that right ankle jerk is absent, and left diminished.

19/8/18 Discharged for evacuation.

Case 3.

Lieut. M., R.E., aged 33, was admitted to the Neurological department on March 23, 1918.

Family history negative. No illnesses in civil life. His first attack of malaria was in June 1917; he had one relapse.

Present illness.

On March 6 he got wet through, and had a slight attack of fever. Next day he experienced a sensation of pins and needles in the left hand. At first slight, it soon became so severe that he could hardly bear to touch arm. He also had a curious itching sensation in the right side of his neck. The sensory disturbance in the hand gradually spread up to the shoulder. On March 15 when he went to bed, he could hardly walk on account of pain in the knees and feet. He was admitted to hospital and had a rigor on March 18. Benign tertian parasites were found, and his spleen was readily palpable. The fever subsided on March 20.

State on admission to Neurological Dept.

Complaint: Pins and needles in upper and lower limbs and in back of neck. Loss of control of left hand.

He is well developed and shows no abnormality of heart or lungs. Spleen enlarged and palpable.

Mental state. Emotional and neurotic. No disorder of memory.

Neurological examination.

Speech and articulation normal. Pupils and cranial nerves normal. No nystagmus.

Sensory functions.

To light touch there is slight blunting in left ulnar distribution. Pinprick evokes a diffuse sensation on left forearm, hand, and fingers. When pricked, the finger tips tingle. There is hyperaesthesia and hyperalgesia over root of neck and shoulder on the left side.

Localisation, joint and kinaesthetic sense are defective in left upper limb. The stereognostic sense is defective in the left hand.

Motor functions.

Right upper limb normal. The left handgrasp is weak; extension at elbow and flexion at wrist are weak. He cannot fully extend left annularis and minimus: there is marked paralysis of the left minimus, and diffuse weakness of the whole limb. There is slight wasting of the left thenar and hypothenar muscles.

There is marked incoordination of the left upper limb in performing finger-nose test.

In taking hold of objects he fumbles with his left hand.

Reflexes: Abdominals present and equal. Plantars, flexion. Arm jerks exaggerated on the left side. knee and ankle jerks brisk.

Lower limbs. The vibrations of a tuning fork are not appreciated; otherwise no sensory loss. The calves are tender to pressure. Motor power normal.

Gait: Base widened; he is unsteady, especially on turning.

Progress

To light touch and pinprick there is blunting on minimus and ulnar side annularis.

29/3/18

Complains of numbness of left hand and fingers. There is now anaesthesia over whole of minimus, and ulnar half annularis; blunting on palmar aspect of other digits.

To pinprick hyperalgesia palmar aspect of all digits of left hand. To pressure marked hyperaesthesia of left digits except minimus. The left ulnar nerve trunk is tender. Vibration sense and joint sense lost at left elbow, wrist, and finger joints.

He cannot perform coordinated movements with the left upper limb or fingers. The ataxia is much increased when the eyes are closed

30/3/18

Electrical reactions: Diminished faradic excitability in the left biceps and in the muscles innervated by the ulnar nerve.

The arm jerks on the left side are now diminished.

2/4/18

There is now blunting to cotton wool touches in the ulnar distribution of the right hand, and hyperalgesia to pinprick over a similar area.

The arm jerks are now equal on the two sides.

11/4/18

Wassermann reaction in blood and cerebrospinal fluid negative

12/4/18

No sensory disturbance in hands now. There is slight motor weakness in the left hand, and the incoordination is much less marked. Gait normal X ray examination negative.

23/4/18

Left upper limb normal except for slight weakness of extensors of minimus and annularis. Flattening of palmar eminence still present.

4/5/18

Complains of indefinite numb sensations in fingers. Motor functions normal

20/5/18

Discharged recovered.

Case 4.

Major D., R.F.A., aged 46, was admitted to the Neurological Department on July 26, 1918.

Family history negative

Previous history. No illnesses of importance in civil life. Temperate. In 1900 he contracted malaria in South Africa. His first attack in Macedonia was in July 1916, and he had numerous slight relapses up to the Spring of 1917. Thereafter he remained free from illness until July 1918.

On July 6 he had a slight attack of malaria, but took no quinine. Two days later he had a very bad attack, the paroxysm lasting eight hours.

Although he felt far from well he went on leave to Athens on July 12. He spent much of his time walking about sight-seeing. On the third day (Aug. 15) his legs felt heavy and tired, and his calf muscles became "cramped". Next day he experienced pins and needles in his feet; this abnormal sensation gradually extended up his limbs, and also invaded his fingers. On August 19 he had anaesthesia of his anus and buttocks, being unconscious of movement of his bowels.

On August 20 he was admitted to a General Hospital, where the following notes were made:-

Slight tenderness on deep pressure in calves. Impairment of sensibility to light touch and pin-prick over arms, legs and buttocks. Rombergism present. Knee and ankle jerks absent.

On August 22 a negative Wassermann reaction in blood was reported.

State on admission to Neurological Dept.

Complaint: Tingling in hands and feet, loss of sensation in buttocks, and inability to walk.

Patient is poorly developed. Pulse 80 per min. Radial arteries slightly thickened. Heart and lungs normal. No enlargement of spleen. Bowels constipated.

Neurological examination.

Higher mental functions: No impairment of perception or memory. Emotional tone one of depression. He sleeps badly at night.

There is a fine tremor of outstretched limbs, and involuntary movements producing alternate flexion and extension in joints of fingers.

Speech and articulation normal. Pupils contracted: reactions normal. Visual fields and visual acuity normal. No nystagmus. Cranial nerves normal.

Sensory functions.

To light cotton wool touches there is blunting of sensibility in the tips of all the fingers of both hands, and a similar condition in the lower extremities, which diminishes in degree in a proximal direction. Above the knees it cannot be demonstrated. He has some difficulty in discriminating between the point and head of a pin on the finger tips, and in a similar situation the threshold for appreciation of simultaneously applied compass points is raised. The vibrations of a tuning fork are felt, but the sensations evoked do not last so long as on a normal area. Localisation normal.

Temperature sense completely abolished in both lower limbs. To pinprick there is hyperaesthesia and paraesthesia on both aspects of the digits. A pinprick on the left index evokes a sensation of a hot wire having been brought into contact with the skin. The hyperaesthesia increases towards the ulnar side of the hand

On the lower limbs there is marked hyperaesthesia up to the groins and gluteal folds, and it is most evident on the toes. Persistence of sensation is marked below the knees.

Joint sense: Upper limbs normal. Lower limbs: there is definite disturbance in the toes, and persistence of the sensations evoked by their passive movement.

There is no tenderness of nerve trunks, but the muscles of both upper limbs are slightly tender, and pressure on the calf muscles is very painful.

Motor functions.

There is no motor paralysis, but both hand grasps are weak; also adduction of fingers towards the middle line. Lower limbs: no loss of power.

There is slight incoordination in performing finger-nose or heel-knee tests. Rombergism is slight.

Gait: He walks on a broadened base, and deviates from side to side; he has especial difficulty in walking backwards, and is worse when his eyes are closed. The calf muscles are hypotonic.

Reflexes: Abdominals brisk and equal; plantars= flexion.

Biceps and supinator jerks absent; triceps jerks present. Knee and ankle jerks absent. Organic reflexes normal.

Electrical reactions.

To faradism there is diminished excitability in glutei and muscles of calves.

Galvanic reactions normal.

Progress.

29/7/18 Rigor at 10 a.m. B.T. parasites present.

2/8/18 Sensory disturbances are now represented by hyperaesthesia to pinprick on limbs up to elbows and knees - with marked persistence of sensation, and impairment of joint sensibility in toes. The muscles are no longer sensitive to pressure.

Motor functions normal. When walking patient has slight difficulty in turming.

There is slight oedema of the feet near the toes.

6/8/18. Gait practically normal. In the earlier s stages of his illness he used to have a feeling as though when lying down his feet were projecting over the end of the bed, due no doubt to the existence of sock anaesthesia.

14/8/18 Slight numbness in little finger; no other sensory disturbance.

17/8/18 Arm and knee jerks are now present

27/8/18 Neuritic symptoms have disappeared. Discharged to duty.

Case 5.

Spr. B., R.O.C., aged 39, was admitted to the Neurological department on October 23, 1918.

Family history negative.

Previous history. No illnesses incivil life; denies venereal disease.

Present Illness.

On October 5 patient got wet through, and next morning found that he could not fasten his braces owing to weakness of his hands. He felt feverish

He was admitted to hospital on 7/10/18 with a temperature of 101.2 F. The following notes were made.

There is paresis of both arms ,more marked in the left. He is unable to make any movement except partial flexion in the left arm Marked wrist drop. He can raise right hand above head; there is a good deal of wasting in the interosseal muscles of the right hand. History of headache, vomiting etc.

State on admission to Neurological Dept.

Patient is a tall, thin subject. Pulse 72 per second. Heart and lungs normal. No enlargement of spleen. Slight anaemia.

Neurological Examination.

Higher mental functions. No impairment of memory, perception or orientation.General intelligence poor.

No tremoe. Speech and articulation normal.

Pupils contracted; react somewhat sluggishly to light and on accomadation. Cranial nerves normal.

Sensory functions.

No cutaneous anaesthesia, hyperaesthesia or analgesia. Temperature sense and localisation normal.

No disturbance of joint sensibility.
Pressure on crural nerves, popliteal nerves, and on muscles of calves elicits pain.
Motor functions.
Range of movement and power in muscles of shoulder girdles is good. Left latissimus dorsi contracts feebly on coughing. Contraction of biceps fair. Marked weakness of triceps on both sides. Supinator longus contracts feebly.
Marked weakness of extensors of wrists, fingers, and thumbs, more obvious on the left side.
Slight weakness of flexors of wrists and fingers. more obvious on the right side.
The flexor longus pollicis is very markedly paralysed, as are also the intrinsic hand muscles on the right side. Patient is unable to hold knife or fork or to perform any fine movements, such as buttoning up his clothes. He is quite unable to touch tip of right minimus or annularis with right thumb, and cannot touch left minimus with left thumb. Signe de Journal present in right hand.
The little finger of both hands is habitually abducted.
There is diffuse wasting of both upper limbs; on both sides there is marked wastinof brachialis anticus,and extensors of wrists and fingers.

There is very marked wasting of the right first dorsal interosseous muscle: a lesser degree on the left side. There is moderate wasting of the other interosseal, thenar,and hypothenar muscles.

Diaphragm and intercostal muscles normal.
Patient cannot sit up nor maintain himself when placed in that posture. There is diffuse weakness of the peronei, long extensors of toes, and of the calf muscles. He cannot stand without support.
Coordination normal. Cerebellar tests normally performed.
Reflexes: Abdominals, upper and lower present, but diminished on right side. Plantars= right, brisk flexion; left= feeble flexion.
Right supinator jerk absent, left diminished.
Right biceps jerk absent, left normal Both triceps jerks absent. Knee jerks present: left greater than right. Right ankle jerk absent: left normal. Organic reflexes normal.
Electrical reactions.
Faradism of right ulnar nerve gives no response. Slight in left ulnar nerve. No response in musculo-spiral nerves. Diminished response in popliteal nerves.
Biceps and pectorals contract briskly: also anterior fibres of deltoid; posterior fibres contract sluggishly. Markedly diminished response in supinator longus of both sides. No faradic excitability in muscles of the right hand: diminished response in those of the left side. The muscles of the lower limbs show diminished faradic excitability.
Galvanism: KCC>ACC all muscles.
Progress
31/10/18 Wassermann reaction in blood positive.

Case 5. Spr. B. Malarial Polyneuritis.
Illustration.

Shows weakness of opposition of the thumb.
Patient is attempting to touch the tips
of his little fingers with the tips of
his thumbs.

6/1/19

There is now diffuse wasting of the muscles of both upper limbs, and in particular in the extensors of the wrists, fingers , and in the intrinsic hand muscles.

He is still unable to stand without support.

7/1/19 Evacuated by hospital ship.

Case 6.

Cpl. B., 2/Cameron Hdrs., aged 25, was admitted to the Neurological department on January 3, 1918.

Family History

Father died of pneumonia;mother of heart disease.

Previous History.

No illnesses in civil life

Present Illness.

About December 1, a few days after a mild attack of malaria, he began to experience pains in the lower part of the legs; his feet had often been cold and damp and he attributed his condition to the wet.

After the lapse of about five days both feet began to feel numb, and he could not walk very far.

State on admission.

Complaint: numbness in feet, tenderness in calves, and weakness of legs.

A healthy looking subject: no enlargement of spleen.

Neurological Examination.

Fine tremor of outstretched limbs. Pupils and cranial nerves normal.

Sensory functions.

To light touch there is blunting on finger tips; On feet there is complete loss in sock distribution, except on right sole. To pinprick there is diminished sensibility in the same areas.
Pressure on right external popliteal nerve, and on calves elicits pain.
Joint sense: upper limbs normal. There is complete loss in the toes of both sides. Heat and cold are not appreciated on the feet. The vibrations of a tuning fork are not felt on the feet or digits, except thumbs.
Motor functions.
Upper limbs normal.
Lower limbs: movements of the toes are slowly and feebly performed.
Gait: patient walks slowly, cautiously, and principally on the heels.
Reflexes: abdominals brisk; plantars absent at toes. Supinator jerks present. Biceps and triceps jerks absent: knee jerks much diminished. Ankle jerks absent.
Patient has areas of hyperidrosis on right eyebrow, tip of nose, upper lip, and chin.
Progress.
9.1.18 States that the ground feels soft beneath his feet. Wassermann reaction was negative on Dec. 26.
10.2.18 Marked improvement. There is now no loss or disturbance of pain, temperature or joint sensibility. There is slight anaesthesia to light touch, and

vibration is not appreciated on toes.

The toes can now be freely moved. The knee jerks, and right ankle jerk are absent: left ankle jerk present.

11.2.18 Transferred for massage and exercises.

Comment.

In Case 1. the sensory and motor disturbances affected principally the muscles of the upper extremities, although weakness of the intercostals, and lower limbs was also observed. It is also to be noted that the affected muscles underwent rapid wasting. Since many of the soldiers of the Royal Serbian Army suffered markedly from malnuitrition, careful enquiry was made with reference to the patient's dietary. As the report was satisfactory, the possibility if the condition being an atypical example of Beri Beri need not be entertained.

The onset in Case 2 was subacute, and the disturbance of sensation unusually marked; in this patient also it is interesting to note that the cranial nerves were not spared: there was a transient facial apralysis, and difficulty in deglutition.

The motor manifestations in Case 3 were very limited, and a casual examination suggested brachial neuritis rather than polyneuritis, but the vibrations of a tuning fork were not felt on the lower limbs, and his gait was markedly ataxic. A rather rare phenomenon was also present, namely an exaggeration of the arm jerks on the affected side, followed at a later date by their diminution.

Case 4 is of interest for several reasons. The polyneuritis was subacute in onset, and accompanied in its initial stages by an anaesthesia

which affected the functions of the rectum, and the sensory defects were the most marked features of the case. Lastly, blood examination showed the presence of benign tertian parasites.

Case 5 was one of the few in which there was also a syphilitic infection, and the very slight improvement recorded suggests that the added syphilitic infection (a positive Wassermann reaction was obtained) militated against the patient's recovery.

It is interesting to note that in this series none of the patients showed the slightest derangement of mental function, although other observors have recorded the Korsakoff syndrome in malarial polyneuritis.

Lastly it may be remarked that only in one instance (Case 1) was there evidence of implication of the heart muscle.

Neuritis.

Although our series of cases is small, malarial neuritis is relatively frequent, and its clinical forms are varied. In the upper limb the most frequent type is a lesion of the posterior branches of the brachial plexus, in which the spinati, and deltoid are particularly involved. In the lower limb it is principally the external popliteal nerve which is attacked, although I have been informed that several cases of lumbosacral neuritis were seen in Macedonia.

Post malarial neuritis begins as a rule with subjective disturbance of sensation, which may persist throughout the whole course of the illness, and dominate the clinical picture. In one case the paralysis appeared suddenly, the patient noticing it when he had recovered consciousness.

Case 1.

Gnr. S., R.F.A., aged 28, was admitted to the Neurological department on September 6, 1917.

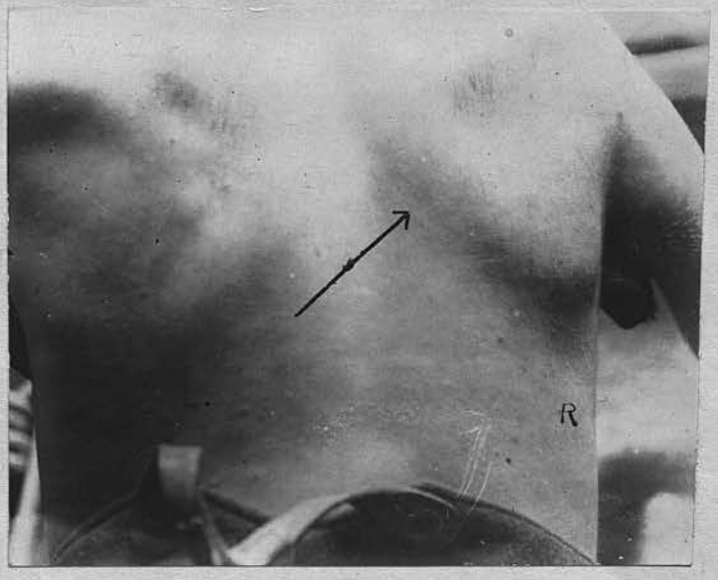
Family history negative.

Previous history.

Scarlet fever in civil life. Sandfly fever in June 1917. First attack of malaria on July 24, 1917. The onset was abrupt and consciousness became clouded for 24 hours. Two days after the cessation of fever(July27) radiating pains of a stabbing and tearing character were experienced in the right upper limb, in the neighbourhood of the shoulder, and posterior aspect of the upper arm. For the relief of pain he required opiates. Seven days

Case 1. Brachial Neuritis.

Note wasting of infraspinatus muscle on right side (indicated by arrow).



later he noticed loss of power in the right upper limb, movements in an upward direction being difficult to perform. The neuritic pains continued to be troublesome until the end of August.
He had no return of fever, and repeated blood examinations were negative.

State on admission.
Patient is thin, undersized, and of poor physique.
Slight anaemia; spleen not enlarged.
Neurological examination.
Mental state normal. Pupils and cranial nerves normal.
Sensory functions.
There is blunting to all forms of sensibility on the right upper limb, from the level of a line encircling the shoulder down to the hand ("hysterical"anaesthesia).
Motor functions.
There is weakness of the right shoulder girdle muscles and of the triceps and biceps on the same side. The right arm can only be abducted a few inches from the side. The right shoulder droops a little, and there is slight wasting of the deltoid and spinati. Passive movements at the shoulder or elbow joints elicit pain
Progress. 17.9.17.
Electrical reactions.
There is diminished excitability to faradism in the anterior half of the right deltoid, and no response in the posterior half.

Galvanism: "R.D." in right supra- and infraspinatus, and in posterior half of deltoid.

The functional anaesthesia has disappeared.

28.9.17.

The right shoulder still droops, and the tip of the shoulder is more prominent than on the normal side. There is obvious wasting of deltoid, spinati, biceps and triceps; also in the middle third of the trapezius

19.19.17

Marked wasting of deltoid and spinati on the right side.

20.19.17

Discharged for electrical treatment and massage.

19.1.18.

Readmitted. There is now diffuse wasting of the right upper limb, and marked wasting of the deltoid and spinati. All movements on the right side are executed freely except abduction and elevation of the upper arm.

Electrical reactions.

Diminished faradic excitability in right biceps, triceps, and extensors of the wrist, fingers, and thumb. The anterior, middle, and posterior segments of the deltoid contract feebly. No reaction in right infraspinatus.

Galvanism ACC>KCC right infraspinatus; KCC>ACC all other muscles.

The right supinator and biceps jerks are not obtained. The right biceps forms no visable swelling when the patient attempts to contract it.

Case 2.

Pte. B., O.B.L.I., aged 32, was admitted to the Neurological Department on October 23, 1917.

Family and previous history negative.

Present illness.

Primary attack of malaria on August 17, 1917. On October 5 he had a relapse; the paroxysm commenced with headache, shivering and vomiting. On the third day there was violent delirium, and on October 10 the parasites of malignant malaria were found in blood film.

On October 15 patient woke in the night with pain in the right shoulder girdle and upper limb. Two days later he noticed weakness, and could not raise the right upper limb above his head.

On October 19 the following notes were made by Col. Purves Stewart:-

"Right arm habitually tonically semiflexed at elbow, and does not swing in walking. To cotton wool touches anaesthesia over whole of right deltoid. To pin-prick less extensive area. Right trapezius excessively feeble: cannot shrug right shoulder. Right deltoid contracts, but not strong enough to produce movement. Triceps and biceps feeble. Latissimus contracts normally on coughing. Wasting right supra- and infraspinatus. Supinator and knee jerks brisk and equal. Tenderness on pressure of right brachial plexus."

State on admission.

Patient is a thin neurotic subject; slight double

exophthalmos. Heart, lungs, thyroid, and spleen normal.

Patient complains of pain in right shoulder and upper arm

Sensory functions.

To light touch there is blunting on an area which extends from ½ inch below acromion process to the middle of the arm. In the same area there is complete analgesia. The following muscles are tender on pressure:n right spinati, trapezius,deltoid,latissimus dorsi, sternomastoid, biceps, triceps, supinator longus, and brachialis anticus.

Motor functions.

There is no voluntary contraction in the right deltoid and trapezius. Tonic contraction still present

Electrical reactions.

Faradic excitability diminished in right deltoid and trapezius. Galvanism: KCC>ACC all muscles.

Progress. 3.11.17

Sensory changes as before.

There is no voluntary power in right deltoid. The right trapezius contracts very feebly. Internal and external rotation at right shoulder joint are feebly performed. There is marked wasting of the deltoid and spinati muscles on the affected side. The tonic flexion of the elbow joint has disappeared.

Tendon reflexes normal. Pressure on the right brachial plexus and musculospiral nerve elicits pain.

12.12.17

Patient is making slow progress and requires con-

Case 2. Pte B. Brachial Neuritis.

Note on right side slight tonic flexion at elbow joint.

stant urging to exercise his paresed limb.

12.1.18

Patient can now hold right upper limb above his head, although he cannot raise it to this position from his side.

24.1.18

Transferred for massage and exercises.

Case 3.

Pte. W., A.S.C., aged 41, was admitted to the Neurological department on November 24, 1917.

Previous illness.

No illness in civil life. Had syphilis in August 1916.

Present illness

On August 27, 1917, he had an attack of malaria of ordinary severity. On September 11, two days after he had resumed duty, he woke to find that he had wrist drop.

State on admission.

A well developed subject; heart, lungs and spleen normal.

Neurological examination.

Somewhat dull mentally. Pupils slightly irregular, and react sluggishly to light. Cranial nerves normal.

Sensory functions.

To light touch complete anaesthesia up to bend of elbow in "sleeve" distribution. Janet's "Yes, NO" test positive. Joint sensibility normal. No tenderness of nerve trunks.

Motor functions.

The right extensors of the wrist, fingers, and thumb are markedly paresed, but no individual movement is impossible. The supinator longus contracts well. He cannot abduct or completely flex fingers of the right hand. There is slight wasting of the extensor muscles of the right forearm.

Electrical reactions.

Diminished faradic excitability in the right supinator longus, triceps, and extensors of the wrist, fingers and thumb.

Reflexes.

The right biceps and supinator jerks cannot be obtained; other reflexes normal.

Progress.

3.12.17 Slightly improved; the hysterical inability to flex wrist and fingers has disappeared.

27.12.17 Still marked wrist drop and paresis of the musculospiral group of the right side.

Electrical reactions. Practically no faradic excitability in the extensors of the right forearm.

On the 19th of January 1918 the patient was brought before a medical board, no further improvement having occurred.

Case 4.

Sgt. R.,Royal Serbian Army, aged 24.

In August and September 1915 he was ill with malaria. Thereafter he remained comparatively well until August 27, 1918, when he had a slight malarial paroxysm. He went to bed on the night of

August 29 feeling fairly well, and woke the following morning with drop wrist. He stated that he slept on his right arm; the limb was in a position of full adduction and supination, flexed acutely at the elbow joint, with his head resting on the ulnar border of the hand.

State on examination.

Complaint:"Pins and needles" on a small area at the base of the right thumb; inability to raise right hand or fingers.

There is no history or evidence of alcoholism.

Sensory functions.

Light touch everywhere appreciated. To pinprick there is blunting over small area on first dorsal interosseous space. Joint and vibration sense normal.

Motor functions.

Right triceps and anconeus normal. Paresis of supinator longus. Complete paralysis of right extensors of wrist, fingers, and thumb.

Reflexes: right supinator jerk diminished: other reflexes normal.

Electrical reactions normal.

Progress

1.9.18. Right supinator longus more paralysed; its jerk now absent.

3.9.18 Faradic excitability of right supinator longus and extensors diminished.

4.9.18 Patient can now extend minimus, and maintain wrist almost extended against gravity

9.9.18. Slight extension of digits of right hand now possible; the wrist can be extended through a considerable angle. Electrical reactions as before. The patient did not report again.

Case 4. Musculospiral paralysis. Patient is attempting to extend both wrists.

Case 5.

Pte T., Royal Serbian Army, aged 41, was admitted to hospital on June 28, 1917.

History.

Ten months ago he had a very severe attack of malaria, with high fever, and unconsciousness lasting five days. When the fever subsided he was found to have paralysis of the left upper extremity.

State on examination. 9.7.17.

His spleen is readily palpable, and B.T. parasites have recently been found in his blood.

Neurological examination.

Pupils and cranial nerves normal.

Sensory functions.

To light touch there is blunting of cutaneous sensibility on the palmar aspect of the left hand in the median distribution, and to a very slight degree in the ulnar area. On the forearm it is possible to distinguish two areas: 1. of very slight blunting on the ulnar aspect. and 2. more marked blunting on the radial surface, including the thumb and index finger.

To pinprick there is a very definite area of diminished sensibility on the dorsal aspect of the hand and thumb. Immediately surrounding this there is a small area which is hyperaesthetic to pinprick. Appreciation of pressure and pressure pain is lost over an almost corresponding area.

The ability to appreciate the vibrations of a tuning fork is impaired on the four radial digits: it is normal on minimus.

Severe pressure on the left musculospiral and ulnar nerves does not elicit pain.

There is no disturbance of sensation on the right upper or both lower extremities.

Motor functions.

Left upper limb. There is complete paralysis of the muscles innervated by the radial nerve (triceps, supinators, extensors of wrist, fingers, and thumb). The hand is in the attitude of wrist drop, and there is a synovial swelling on the dorsal aspect of the carpus. The other muscles of the left upper limb, and those of the shoulder girdle show a moderate degree of paresis - notably the deltoid and biceps. The handgrasp is very weak, and the strength of the muscles innervated by the circumflex, musculocutaneous, median, and ulnar nerves is much less than on the normal right side.

There is very marked wasting of triceps, supinators, and extensors of wrist, fingers, and thumb; considerable wasting of biceps and triceps.

The circumference of the forearm 10cm. below the olecranon process is 22cm. On the right side it is 27cm.

Reflexes. The left supinator jerk is absent; biceps jerk present: triceps jerk inverted. Arm jerks normal on the right side. Knee jerks are much exaggerated. Plantars= flexion.

Trophic functions.

The left hand is obviously smaller than the right: the diminution in size is very apparent in the thumb.

The skin of the left hand is pink in colour, and of a fine texture. The finger tips are marked by vertical lines, and the nails are more curved than those on the right side. There is an abnormal growth of hair on the hand and forearm.

X ray examination shows that there is atrophy of the bones of the left hand, which is most marked in the phalanges.

The motor functions of the lower limbs are normal.

Illustration 1.

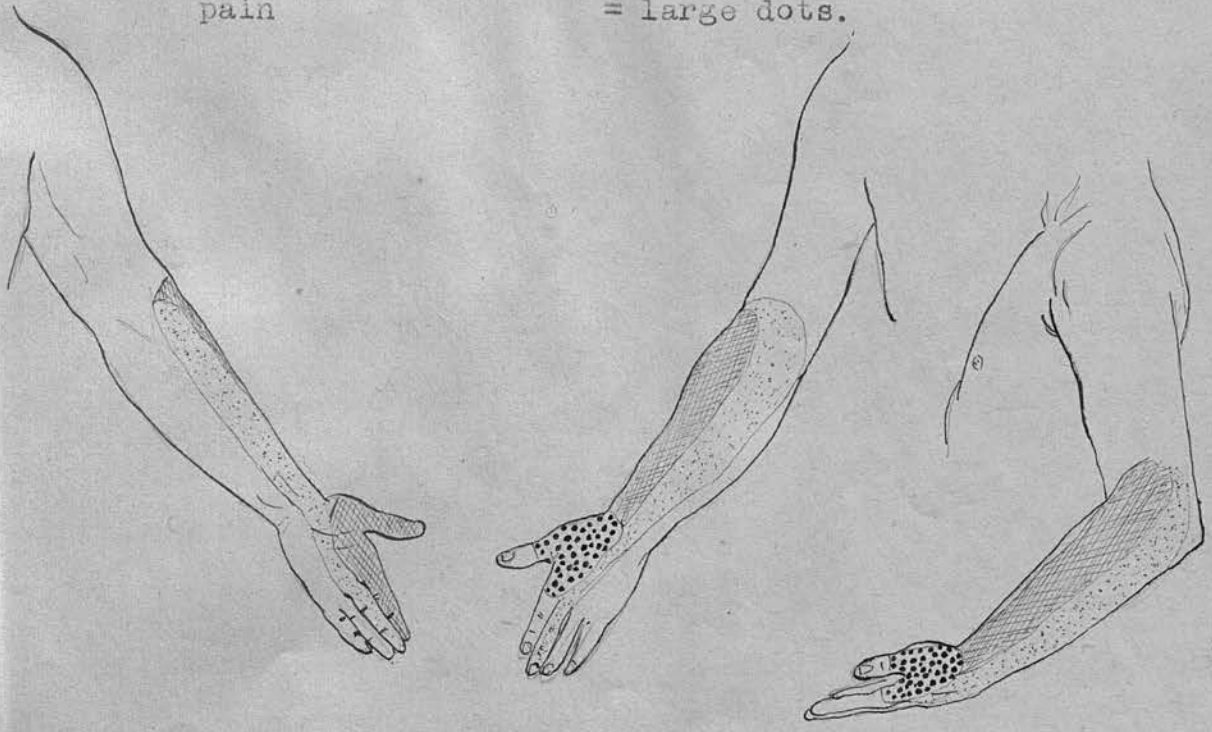
Patient is attempting to abduct fully both upper limbs, with wrists extended.
Note wrist drop, and diffuse wasting on the left side.

Case 5.

Illustration 2. To show trophic changes in the left hand. The left thumb is smaller than the right, the fingers are tapered, the nails curved, and the lines on the skin finer than on the normal hand.

Chart of the sensory disturbance.

slight hypo-aesthesia = fine dots.
marked blunting to pain, pressure, & pressure pain = large dots.



Case 6. Cervical Neuritis.

Pte. M., 23/Welsh Rgt., aged 34, was admitted to the Neurological department on July 26, 1918.

Family history negative. Scarlet fever in civil life.

Present Illness.

Patient contracted malaria in August 1916, and had five subsequent attacks.

In March 1918 the patient began to suffer at night from pain in the right shoulder, and ribs on the same side. A few weeks later he had another attack of malaria, which aggrevated the condition. He was admitted to a general hospital on May 30.

State on admission.

Pupils and cranial nerves normal. Sensory functions normal.

Motor functions.

There is paresis of the right trapezius. He can only raise shoulder to a slight extent on the right side and has difficulty in raising his arm above the horizontal. When both upper limbs are extended in front of him, the lower angle of the right scapula becomes winged. The tip of the right shoulder is lower than the left, and the lower angle of the right scapula is higher and nearer the middle line than the left.

Electrical reactions. There is diminished faradic excitability in the right trapezius and supraspinatus.

Reflexes The right supinator and biceps jerks are diminished; other reflexes normal.

Case 7.

Sgnlr. T.H., 2/Cheshire Rgt., aged 22, was admitted to the Neurological Department on August 24, 1918.

Family History. Mother suffers from a nervous disease.

Previous History.

No serious illnesses in civil life.

On August 4, 1915 patient was vaccinated on the left arm. A severe cellulitis, followed by ulceration ensued, and patient was thirteen months under treatment, and had considerable stiffness of the shoulder and elbow joints. Eventually the left upper limb became almost as strong as the right, although the muscles on that side looked smaller than normal.

Present illness.

Patient had a primary attack of malaria in June, 1917, and had four subsequent relapses. One week after the last attack, while he was in a convalescent camp he experienced a dull, aching pain in the left upper extremity, aggrevated by physical exercise. The limb also felt weak. He was admitted to hospital with another attack of malaria, and after a month's treatment transferred to the Neurological department.

State on admission.

Complaint: Weakness of the left arm.

Patient is a thin anaemic subject. No abnormality of heart, lungs or spleen. Urine normal.

Higher mental functions normal. Pupils and cranila nerves normal.

Sensory functions.

No cutaneous anaesthesia or anaesthesia. Joint sense normal. Pressure on the left biceps, triceps, brachialis anticus,and musculospiral nerve elicits pain. Motor functions: Normal except in the left upper limb.

There is a moderate degree of paresis in the left deltoid, biceps, brachialis anticus, and triceps. There is wasting of these muscles, the biceps being particularly affected. The circumference of the upper arm 10 cm. below the tip of the acromion is 26 cm. on the right, 22.5 cm. on the left limb. Abductionof the left upper limb to the horizontal, and flexion and extension of the left elbow are less powerfully performed than on the right side. Reflexes: Abdominals brisk and equal; plantars= flexion. The arm jerks are present but brisker on the left side than on the right. The knee and ankle jerks are brisk and equal.

On the outer side of the left upper arm there are three large cicatrices, pink in colour, due apparently to ulceration following vaccination.

Electrical reactions.

There is diminished faradic excitability of the left deltoid biceps, and triceps. Galvanic reactions normal.

Progress

Under appropriate treatment the sensory (subjective) disturbance disappeared by September 5. The motor defect remained. On September 11 the patient had a very severe attack of malaria, with grave abdominal symptoms, which necessitated his early evacuation.

Comment.

Cases 1 and 2 showed certain points of resemblance in that in both, the deltoid and spinati muscles were markedly affected. In the second case in which there was a history of syphilis, the degree of improvement after several months energetic treatment was very slight. Attention has already been drawn to a similar delay in recovery in a case of polyneuritis in which there was also a history of syphilitic infection.

In case 4, an example of very transient musculospiral paralysis, pressure undoubtedly played an important part in determining the condition, but as in other cases, there appeared to be an additional factor - malaria. It might therefore be said to be toxico-traumatic in origin.

Case 5 was remarkable for the very severe degree of paralysis, the patient's left upper extremity suggesting at first glance, a limb affected by an attack of poliomyelitis.

In Case 7 it is probable that the cellulitis and ulceration, which had occurred some years previous to the malarial infection, had caused slight degeneration of the peripheral nerves, predisposing them to the influence of new toxins.

Herpes Zoster.

Herpes labialis is exceedingly common in malaria, and usually makes its appearance during the stage of pyrexia. As in other febrile states, it is probably of infective origin, and has to be distinguished from true herpes zoster, which is totally different in its clinical course and pathology.

In only one instance was it possible to trace a relationship between malaria and zona.

Dvr. B., B.A.C., aged 22, was admitted to hospital on August 14, 1918.

His first attack of malaria was in July 1916, and he had more than twenty subsequent relapses.

On admission to hospital he had the usual symptoms of a mild attack of fever, his temperature remaining elevated until August 26.

On August 21 pain in the back made its appearance; it was of great severity, as though a knife was sticking into his back. Three days later a typical herpetic eruption commenced. At first it was visable only behind, but it soon spread round to the front of the chest.

Distribution of eruption.

Vesicles are present over the left scapular area, and to a slight extent they cross the middle line. They also extend on to the posterior fold of the axilla, and inner border of the upper arm. In front there is a small patch three inches internal to and a little above the nipple.

There are a few scattered vesicles on the anterior fold of the axilla.

Their distribution appears to be in the territory of the second and third posterior nerve roots.

Illustration 1. Herpes Zoster; anterior view.

Illustration 2. Herpes Zoster; posterior view.

Malarial Tremor

Among the admissions to the Neurological Department tremor was observed in a large percentage. In many cases it appeared to be functional in character, being especially common in the War Neurosis group; in others it seemed to owe its origin to excessive cigarette smoking, while in a few it was related to the "Effort Syndrome" or to hyperthyroidism. Nevertheless, when these causes were excluded there remained a large number in whom malaria appeared to be the causal factor, for on examination of a large number of patients undergoing treatment in the Malaria wards numerous instances of tremor were found, especially in those who gave histories of repeated attacks.

The toxic tremor of malaria showed the following characters:-

It was bilateral, involving the upper extremities to an equal extent; the lower limbs were seldom affected, but the tongue often participated.

During rest there was very little tremor, but it became exaggerated as the result of effort, and might even be visable in the handwriting.

The oscillations were rapid - about 8 per second- and were slightly irregular in amplitude.

It was difficult to determine at what period of the disease the tremor appeared, but in not a few cases it dated from the onset of the paroxysm. Repeated febrile attacks seemed to exaggerate the condition, and the benifit derived from treatment was slight.

Trophic Disturbances.

No cases were admitted to the Neurological department on account of trophic disturbances, but the writer can recall several cases among Serbian soldiers of peripheral circulatory disorder.

Perhaps the commonest was a condition of acrocyanosis, with a varying degree of lividity, and sometimes oedema. It was usually seen in the upper extremities, and was often accompanied by subjective and objective feelings of cold.

Instances of Raynaud's disease of malarial origin have also been reported (vide Paisseau), and Samaja,(23) who reviewed this subject, concluded that symmetrical gangrene of malarial origin may precede, accompany, or follow the paroxysm. Cold appears to be an almost indispensable factor in its production, but in his case gangrene of the extremities appeared in August, and was cured by quinine in November.

Oedema of the lower extremities has also been met with. It is probably of vasomotor origin, since no cardiac or renal disturbances have been found.

Lastly, attention has been drawn by Saouda(24) to malarial erythema. This cutaneous manifestation often appears in successive crops, lasting from three to four weeks. It covers a large area of the body, but usually spares the face, neck, and soles. Being often accompanied by Sergent's white line, Saouda concludes that it is due to vasomotor disturbance of trophic origin.

Pathology.

Laveran's discovery of the Haematozoon malariae gave rise to what may be termed the mechanical theory of the lesions characteristic of the disease. The parasites, living within the red blood corpuscles, bring about a destruction of the same, with a liberation of their spores, and the haematoidin of the destroyed corpuscles. It was therefore thought that pigment masses, by blockage of capillary vessels, arrested the circulation in minute areas, and so led to fibrotic and functional disturbances. It appears more probable, however, that under certain conditions the spores of the parasites are liable to become adherent, one to the other, forming with their attached pigment relatively large masses which act as capillary emboli.

This modified theory is widely held at the present time, and is accepted as the explanation of the sequence of events observed not only in cerebral malaria, but in other nervous complications as well. Nor can it be doubted that such changes do often occur. I have myself observed blocking of small vessels in the brain by parasites and masses of pigment, with circumscribed nodules of neuroglial proliferation, but there are certain considerations which militate against the acceptance of this hypothesis as an explanation of <u>all</u> nervous sequelae.

First, mechanical blockage of capillaries may not be found even after the most prolonged search

by Experts in the Base Laboratory.

Manson-Bahr in a report on Malaria in the Egyptian Expeditionary Force (25) states:-

" The first point to note is the evidence of a malaria toxaemia as against the hitherto accepted theory of a mechanical obstruction of the capillaries by the malignant parasite. For instance, no such obstruction could be substantiated in cases with cerebral involvement after quinine treatment."

Secondly, the prompt return of consciousness which not infrequently follows the intravenous injection of quinine suggests that the mechanical theory cannot be considered adequate, for it is difficult to conceive how such treatment could restore a capillary circulation which has been blocked by spores and masses of pigment.

Still more difficult is it to reconcile this theory with many of the clinical manifestations described above. Cases are on record of malarial paralysis strictly hemiplegic in distribution. Are we in such cases to assume that there occurred capillary blockage strictly limited to one cerebral hemisphere ?

The fact that so many cases were examples of neuritis and polyneuritis is of great significance, and makes it clear that malaria acts in a toxic manner upon the whole nervous system, now at this site, now at that, but especially on the cranial and peripheral nerves.

It would appear then, that the essential basis of many of the nervous complications of malaria, be they, cerebral, cerebellar, or peripheral, is a toxic rather than a mechanical one.

It therefore remains to inquire into the nature of the toxic agent. Several possibilities must be entertained.

1. Haematogenous infection of the nervous system by a toxin liberated by the parasite. So far as I am aware no proof in favour of this hypothesis has yet been brought forward, although there is much to be said in its favour.

2. A product of disturbance of the normal kidney function. According to this theory malaria induces a condition analagous to uraemia, which in turn determines the various cerebral and other nervous perturbations. Apart from the striking degree of oedema of the brain which may be seen in fatal cases there does not appear to be any foundation for such a hypothesis.

3. It has been urged that a failure of adrenalin secretion, brought about by a malarial inflammation of the suprarenal bodies, in some way determines the symptoms.

Paisseau and Lemaire describe degenerative and haemorrhagic lesions of the adrenal capsules in patients succumbing to malaria, and correlate the clinical features with the adrenal insufficiency. While it is possible that certain symptoms - the

profound asthenia, the arterial hypotension, the profuse vomiting and diarrhoea - may be an expression of a disturbance of suprarenal secretion, it is by no means easy to understand how such a condition could determine a great variety of nervous syndromes. Dudgeon and Clarke[27], who examined the suprarenal glands in 35 patients dying of malaria, conclude that suprarenal insufficiency does not play the important role which has been ascribed to it. They also found fatty degeneration of the heart muscle in 23 out of 45 fatal cases, which suggests (on the analogy of myocardial degeneration in diphtheria) a toxic origin.

Further views might be given, but enough has been said to show that there is little unanimity of opinion as to the nature of the supposed malaria toxin.

Treatment.

Treatment of the nervous complications of malaria may be discussed from two aspects:-

1. Treatment of the general condition of the patient.
2. Treatment of the nervous complication.

1.

Since treatment is necessarily determined by the special requirements of individual cases, no more is attempted in the following remarks than to summarise the general lines along which it was carried out.

In all cases where there were clinical grounds for suspecting a malarial infection, the daily administration of quinine was adopted at as early a stage as possible.

As a routine measure 15 gr. of the sulphate of quinine were given by the mouth three times a day until convalescence was established. This was always preceded by an initial dose of calomel, followed by a saline aperient. When the fever subsided a smaller dosage was employed.

In cases presenting urgent symptoms resort was had to intramuscular injections of the bihydrochloride of quinine. I was never able to satisfy myself that this form of treatment was more effective and speedy in its effects than the oral method of administration. The results from faulty technique, or from accidental injury to nerve trunks, were in many cases lamentable, and quite a number of cases of deltoid or sciatic

paralysis were admitted to the Neurological department. They were peculiarly intractible, and on account of the severe subjective pains, entailed much suffering.

Cases presenting symptoms of cerebral malaria, or of involvement of the central nervous system, were treated by the prompt administration of quinine intravenously, with gratifying results in many instances. It might even be said of quinine that in the treatment of nervous complications, it proved of a value not much inferior to that of salvarsan in neuro-syphilis.

2.

Lumbar puncture was performed in a certain number of cases, and while it yielded additional information of diagnostic importance, it did not appear to effect any improvement in the symptoms, as has been suggested by certain writers.

It was in the cases which were examples of neuritis or polyneuritis that the ordinary therapeutic agents of the neurologist appeared to be of special value.

In the treatment of facial paralysis, in addition to the routine measures, such as applying blisters and giving diaphoretics, ionisation was carried out daily. The salt employed on the negative pole was usually salicylate of soda, and good results were obtained. Facial "splints"were also used.

In those muscles whose faradic excitability was preserved, the faradic current was employed, gal-

vanism being reserved for nerves exhibiting reactions of degeneration. Massage was found a useful adjuvant.

The oral administration of the liquor of strychnine hydrochloride in doses increasing to 45 minims seemed to prove of value, especially in cases of multiple neuritis.

Conclusions.

1. The parasites of malaria attack with relative frequency the nervous tissues.

2. The nervous complications of malaria are very varied in their clinical forms.

3. Unlike certain other intoxications (e.g. lead and alcohol) they are not subject to any law as regards their distribution.

4. The nervous system may be attacked at any of its levels.

5. A study of the cases in this series, and a review of the literature relating to malaria permits the following classification of the nervous complications.

1. Diffuse cerebral affections, including malial Meningitis.
   - A. "pernicious" cerebral Malaria
   - B. "Benign cerebral Malaria

2. Focal cerebral affections.
   - A. Pseudobulbar paralysis
   - B. Hemiplegia
   - C. Monoplegia

3. Cerebellar affections.

4. Cranial nerve palsies.

5. Myelomalacia, acute or subacute.

6. Polyneuritis

7. Neuritis

A. Cervical

B. Brachial

C. Musculospiral

D. Crural

8. Herpes Zoster

9. Tremor

10. Trophic Affections.

6. While it is in the malignant types of fever that nervous complications are to be expected, benign tertian fever may also be associated with affections of any part of the nervous system.

7. The theory of mechanical blockage of capillaries by parasites, pigment, and proliferated cells, cannot be accepted as an adequate explanation of the numerous and diverse nervous sequelae observed in malaria. Clinical data indicate, though not conclusively, that the malaria parasite secretes a toxin capable of exercising harmful effects not only on the nervous system, but upon all the organs of the body.

8. The prognosis in the cerebral and in the spinal complications of malaria is always grave; in other nervous affections it is favourable.

9. The administration of quinine is of great therapeutic value in the nervous complications of malaria, but an antecedent syphilitic infection has a retarding influence upon the effects of treatment.

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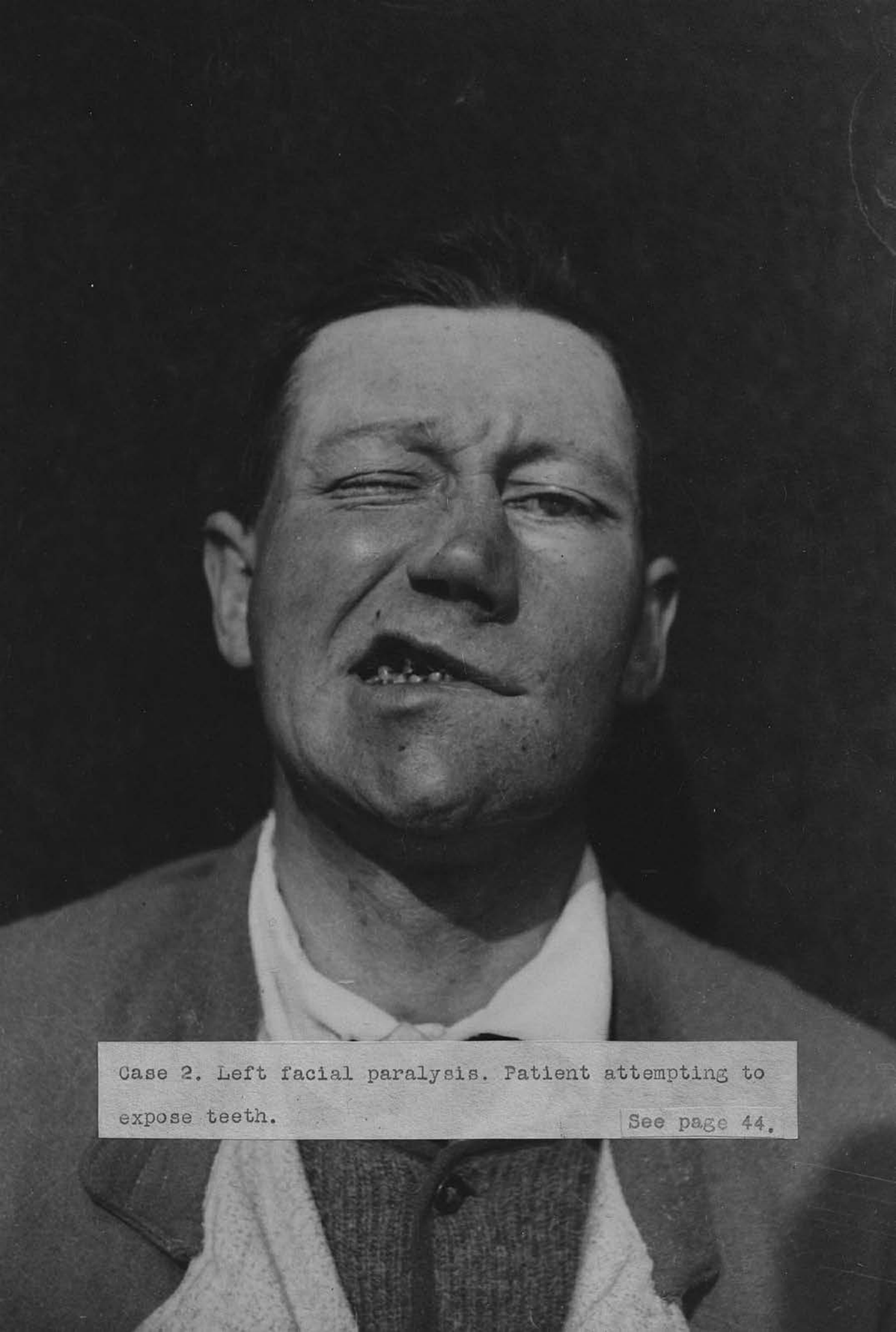
See page 44.

Case 2. Left facial paralysis. Face at rest.

Note widely open eye, deviation of tip of nose, and drooping of mouth on affected side.

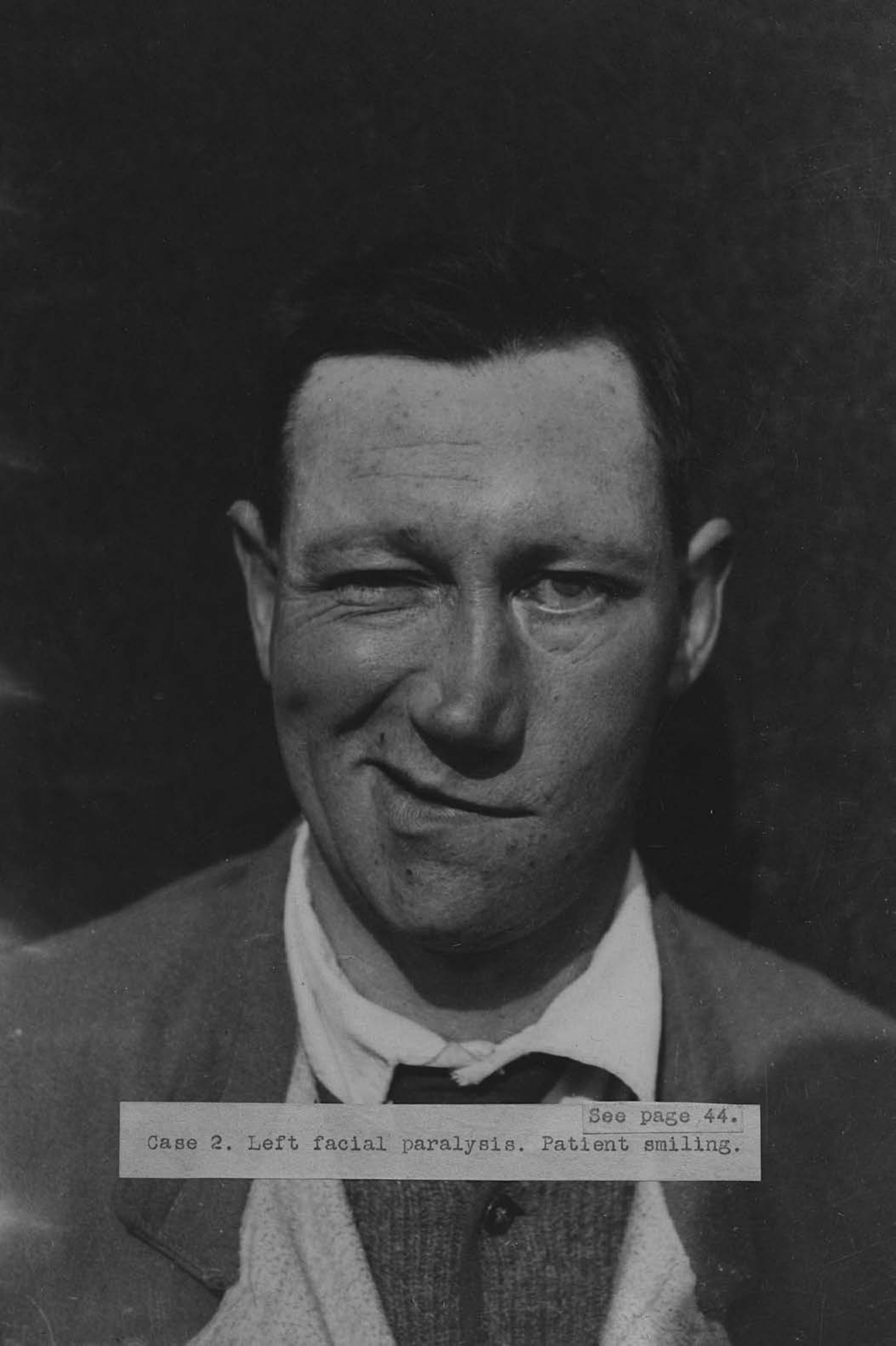


Case 2. Left facial paralysis. Patient attempting to expose teeth.

See page 44.

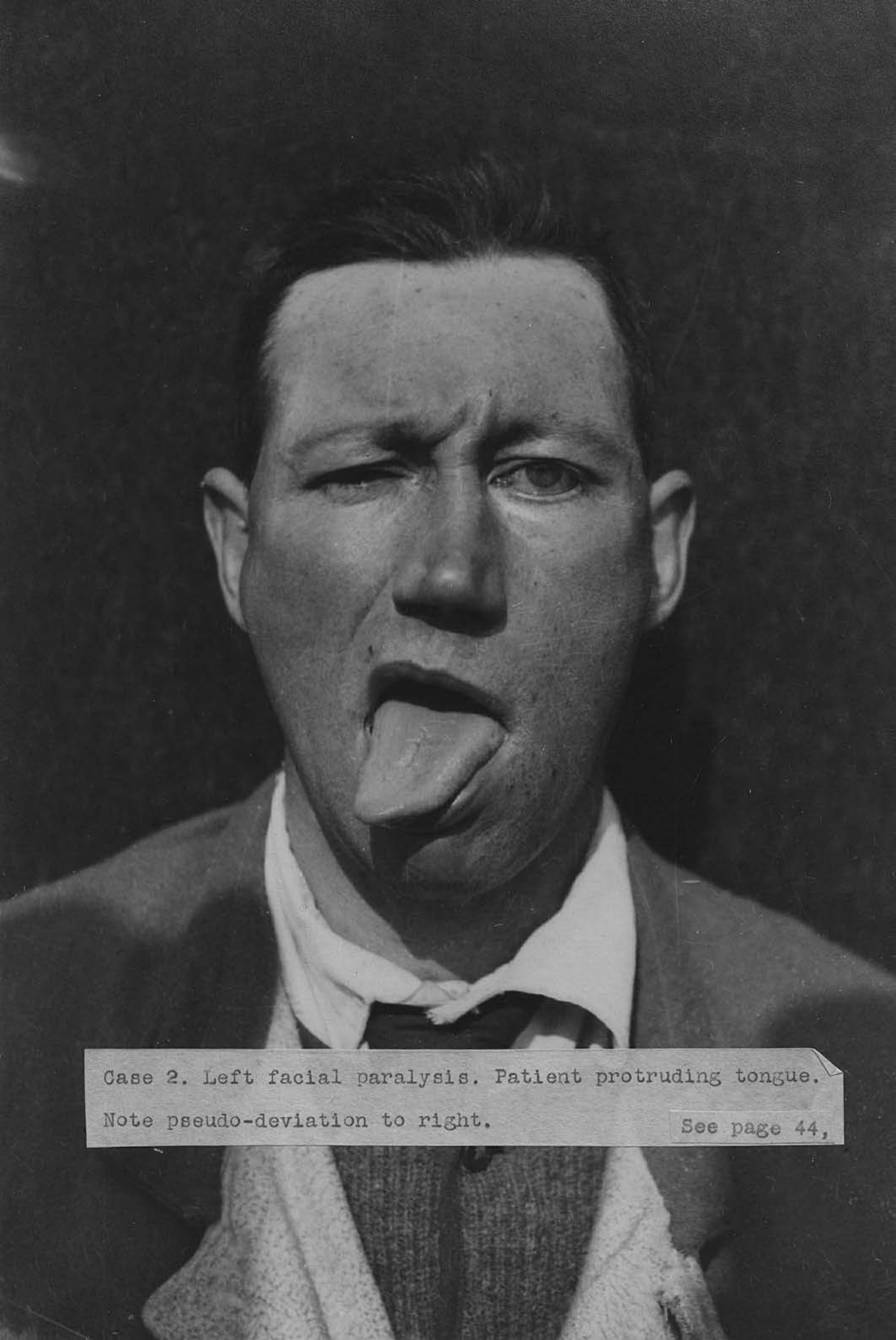
See page 44.

Case 2. Left facial paralysis. Forcible closure of eyes. Note almost entire absence of movement on left side.



See page 44.

Case 2. Left facial paralysis. Patient smiling.



Case 2. Left facial paralysis. Patient protruding tongue. Note pseudo-deviation to right.

See page 44,